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THE UVULA

Its Structure and Function and Its Importance

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AND
E. MARKEY PULLEN, M.D.
NEW YORK

LEDERER'S [1] text offers as clear and concise a picture as may be found of the afflictions of the uvula and the soft palate. On the whole, there is no structure within the confines of the body so little understood and so often abused and maligned as the uvula. This structure's function appears in the literature in only the briefest fashion. The uvula has been classed with vestigial structures, such as the vermiform appendix.

Recently it was announced in the press that in 1946 the scheduled American airlines carried fourteen million passengers a total of seven billion miles. This announcement not only foretold man's changing life but assured one of the ever-increasing importance of the lateral wall of the nose and the nasopharynx together with the middle ear, in the comfort of millions of air travelers.

It is not our intent in this paper to go into the dangers of curetting about the torus tubarius in adenoidectomy, or into the changes that occur in the closed or diseased cavities of the nose and the ear with altitudinal and barometric change. The otolaryngologist of fifty or a hundred years hence may find that his entire practice has become concerned with sinus ostiums and the eustachian tube. We turn our attention to the aforementioned little understood midline structure, in regard to which no little difficulty is encountered in ascribing a function. We present certain of our beliefs and support them with observations, outlining only the cases that are very typical of those seen in persons of any age.

RELATED ANATOMY: NOSE, SOFT PALATE AND UVULA (FIGS. 1 AND 2)

Nose.—Robinson [2] stated that "the pars nasalis of the pharynx differs from the rest of the pharynx in that its cavity remains, under all con-

1. Lederer, F. L.: Diseases of Ear, Nose and Throat, ed. 4, Philadelphia, F. A. Davis Company, 1943, p. 532.

2. Cunningham, D. J.: Text Book of Anatomy, edited by A. Robinson, ed. 6, New York, William Wood & Company, 1931.

ditions, a distinct chamber incapable of obliteration since all its walls, with the single exception of the floor, are practically immovable."

The margins of the posterior choanae narium form the line of separation between the pars nasalis and the cavity of the nose and are occasionally marked by a furrow or a fold on the side wall called the nasopharyngeal furrow. The lower end of this furrow is directed toward the midline.

Probably one of the structures most important to otolaryngologists appears next on the lateral wall. The levator veli palatini muscle runs parallel to the eustachian tube and along its lower border. As it enters the palate it produces, particularly when contracted, an elevation just below the pharyngeal orifice known as the torus levatorius. This and the rounded cartilaginous ridge of the auditory tube known as the torus tubarius serve also as dikes to keep as much material as possible from flowing directly across the eustachian orifice.

Brash and Jamieson [3] stated that the soft palate is the only wall of the nasopharynx capable of movement.

In the midline of the posterior pharyngeal wall, hidden from the view of the observer, is a mass of lymphoid tissue known as the adenoid. One of several collections of lymphoid tissue in the region, it is the only one to assume importance in this discussion. It is of some size in the young; it may atrophy, and not uncommonly it produces fibrotic pockets to retain infected material at the posterosuperior wall, and is commonly subject to surgical removal.

Superior to this structure and appearing after the age of 8 are the two paired sinuses of the sphenoid bone. These have variously placed ostiums and are devoid of cilia, so that material must be extruded over the vault of the pharynx or must travel down the posterior septal margin. Proetz [4] noted that large masses may emerge from the sinuses, assume globular shape in the nose and fall back into the pharynx to the great consternation of the patient. This phenomenon has been observed in local tonsillectomy, and the mass gravitates downward without much difficulty.

The movement of the ciliary stream from the remaining accessory sinuses is always toward the pharynx and oropharynx, across various portions of the lateral wall but around the eustachian orifice to appear on the lateral wall of the pharynx. The detailed pathways for each sinus are excellently described by Proetz in his "Essays."

Secretions accumulating in the floor of the nose during acute infections of the upper respiratory tract, with the ciliary stream slowed by the viscosity and the density of the secretion, are commonly removed

3. Cunningham, D. J.: Manual of Practical Anatomy, edited by J. C. Brash and E. B. Jamieson, ed. 9, London, Oxford University Press, 1935, vol. 3, p. 287.

4. Proetz, A. W.: Essays on the Applied Physiology of the Nose. St. Louis, Annals Publishing Company, 1941, p. 65.

by one of two means—blowing the nose or "hawking." The latter procedure is accepted as the greater aid to the ciliary stream in that work of the cilia is not repeated. On the creation of negative pressure these secretions appear in the mouth for expectoration.

Such secretion, in traveling this pathway during sleeping hours, may move backward and dam up, so to speak, in the posterior regions of the nose or be partly retained in the adenoidal pockets previously

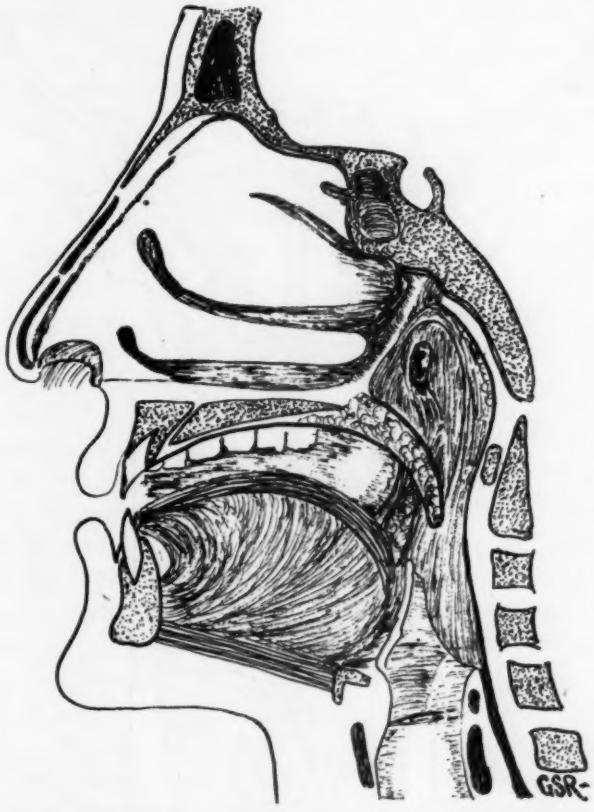

Fig. 1.—Sagittal section through the mouth, the tongue, the larynx, the pharynx and the nasal cavity. The posterior edge of the nasal septum is shown and hides the nasopharyngeal furrow. Also shown are the right sphenoid sinus, the right lateral wall of the pars nasalis, the eustachian orifice with the torus tubarius and the torus lavatorius and the pharyngeal recess above the torus tubarius. The uvula is represented as the posterior projection of the soft palate. (After Cunningham,[2] page 1135, figure 893.)

mentioned. The secretions are further subjected to the drying effect of mouth breathing, to coat the posterior pharyngeal wall below the velum of the palate. The lower margin appears quite dry and is responsible in part for certain symptoms experienced by any one so affected. The attempts of the patient to loosen this material on awakening are respon-

sible for considerable noise. The noise is made within the cavities of the nose on inspiration and by the larynx and the uvula on expiration prior to expectoration. This flutter of the uvula is more than an incidental finding; it is a means of loosening the secretion on the posterior pharyngeal wall.

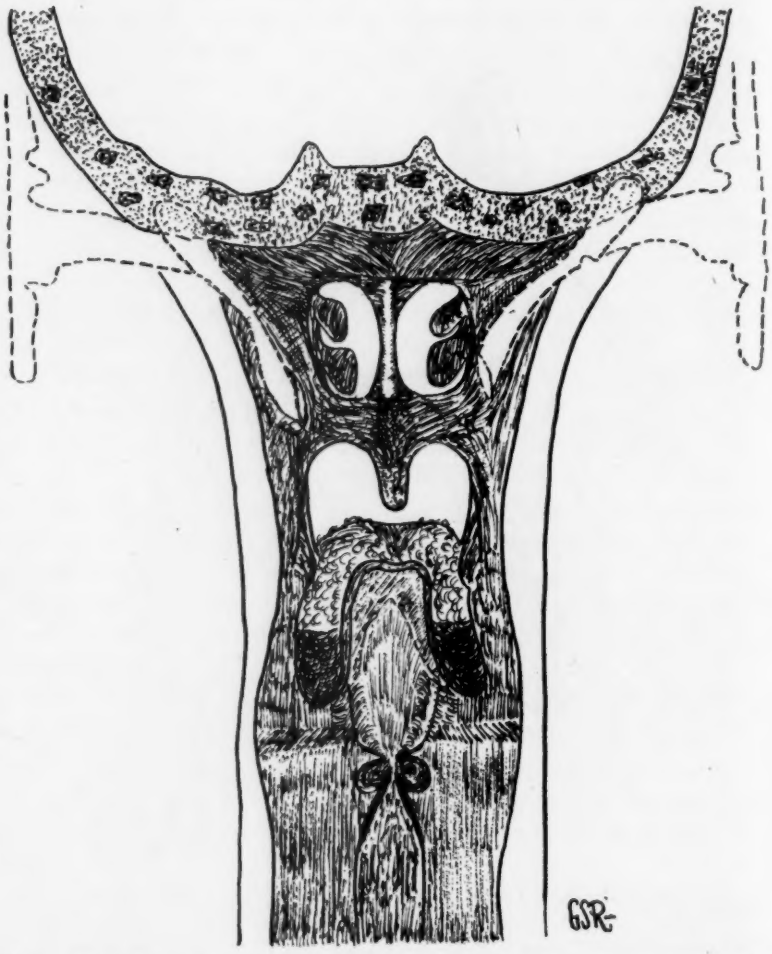

Fig. 2.—Interior of the pharynx and the choanae narium viewed from behind. The approximate relation of the eustachian orifice and the inferior turbinate is schematically outlined on each side, as is the tube and the middle and external ear.

Moisture is secreted throughout the turbinal structures of the nose to humidify the inspired air. When a secretion becomes so viscid and dry as to move slowly, the glands of the palate and the uvula are the only structures capable of altering the viscosity of the secretion moving toward the oropharynx, but in particular that of the lower partially dried and advancing margin.

Soft Palate and Uvula.—Anatomists have described the soft palate as a backward projection of two layers of mucous membrane from the hard palate, enclosing muscles, an aponeurosis, racemose glands above the lower layer, arteries, nerves, lymph vessels and veins.

From the posterior and free margin of the soft palate projects a conical process known as the uvula. It is composed chiefly of a mass of racemose glands and connective tissue, covered by mucous membrane and containing slender prolongations of the musculus uvulae in its upper, contractile part.

The musculus uvulae consists of a pair of slender slips that lie side by side under cover of the superficial fibers of the pharyngopalatinus muscle. They arise from the posterior nasal spine, pass backward through the soft palate, unite and enter the uvula to be inserted into its mucous membrane. They shorten the uvula and are supplied by the accessory nerve through the pharyngeal plexus. A uvula continuously deviated to the right or to the left indicates a weakness, therefore, of the opposite side.

The remaining muscles of the soft palate are the levator veli palatini, the tensor veli palatini, the glossopalatinus and the pharyngopalatinus. The paired glossopalatini form the anterior pillars, while the pharyngopalatini form the posterior pillars. All these muscles are supplied by the accessory nerve through the pharyngeal plexus (motor root joining the vagus nerve) except the tensor veli palatini, which is supplied by the mandibular nerve from the otic ganglion. The ganglion receives possibly sensory and secretory fibers by the lesser superficial petrosal nerve from the facial and glossopharyngeal nerves.

The tensor veli palatini is probably the chief means of opening the pharyngeal orifice of the eustachian tube on either side. By virtue of the innervation, both orifices are opened simultaneously.

The arteries of the soft palate are the ascending palatine (from the facial artery), the palatine branch from the ascending pharyngeal artery and twigs from the greater palatine of the internal maxillary artery. The nerves are the lesser palatine and branches from the pharyngeal plexus.

The uvula is supplied by parent arterial branches from the soft palate, while sensory nerve fibers run to the uvula from the sphenopalatine ganglion through the middle or external palatine nerve.

The midline structures of the nasopharynx are the posterior border of the septum and the uvula with the midline structure on the posterior pharyngeal wall—the adenoid—and this may or may not be present.

The upper surface of the soft palate is described as convex and continuous with the floor of the nasal cavities. The inferior surface is concave and forms part of the vaulted roof of the mouth. We believe

that these commonly used descriptive terms are true in the cadaveric state or during swallowing but that the reverse is true to a limited extent in the relaxed living state. The velum, as shown in figure 3, may descend to the level of the back of the tongue.

During deglutition the two palatopharyngeal arches move up and close the nasopharynx from the oropharynx. The uvula enters into this process but is of importance only as a central reenforcement of the closure. If a person with normal or nearly normal palatine arches is observed as he breathes through the nose with mouth open, the central portion of each arch is seen to move upward and toward the midline. With expiration and relaxation one gains the impression that the midline relaxes before either of the two arches.

The glossopalatini (anterior pillars) arise from the base of the uvula and pass downward to join the side of the tongue a little behind

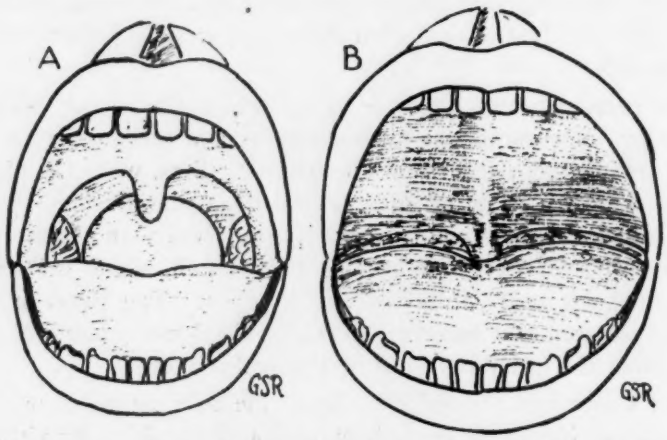

Fig. 3.—*A*, open mouth showing the palate and the palatine tonsils. Also shown are the palatine or pharyngeal arches with the nasopharyngeal isthmus. The uvula is in the midline, and both it and the palate appear much higher than in the cadaver. The philtrum is above. (After Cunningham,[2] page 1105, figure 866.)

B, approximate position of the uvula in a relaxed, living, erect person who was breathing through the nose with the mouth open. This person had been tonsillectomized.

its middle. These are the only extrinsic muscles of the tongue belonging to muscles of the soft palate which are intimately associated with the tongue to aid in altering its position and shape.

The pharyngopalatini spring from the posterior edge of the soft palate and pass downward to end in the side of the pharynx. These and the glossopalatini may narrow the isthmus. The normal uvula projects more from the anterior than the free posterior margin of the soft palate.

When the uvula and the soft palate are elevated by the tensor veli palatini (stretching between the two medial pterygoid laminas), the levator veli palatini and the musculus uvulae, and the pharynx is nar-

rowed by the superior constrictor the whole nasopharynx is closed off from the oropharynx. All of these structures are inextricably woven into one fabric.

This entire process, together with the preservation of structures in the midline or closely approximating it, assumes great importance in the preservation of nasopharyngeal normalcy. It is not uncommon to find that a unilateral aural discharge is associated with a vomerine septal spur in the posterior reaches of the nose, while the opposite ear is unaffected. Secretions have been directed toward the eustachian orifice on the affected side and away from the midline.

In addition to tinnitus and obstructive deafness occurring in similar conditions from faulty inflation, observed by most otolaryngologists, Johnson [5] offers audiometric studies in cases of nasal obstruction. These further emphasize the importance of midline structures in preserving nasopharyngeal and otologic normalcy.

The Epithelium.—It is a basic premise that the character of the epithelium is influenced throughout by the air as it streams through the nose and the nasal cavities. In broad terms, the mucosa changes from a stratified squamous epithelium in the nasal vestibule to a pseudostratified columnar to ciliated columnar epithelium on the lateral wall of the nose and the pharynx and again becomes stratified squamous epithelium on the posterior pharyngeal wall below the posterior choanae. But, wherever the ciliated epithelium becomes directly buffeted by air, it reverts to the stratified squamous type. This has happened on the anterior tips of the inferior turbinates, the upper anterior part of the middle turbinate and the septal tubercle.

Proetz [6] observed "whether by design or by accident, and there is little of the latter in nature, the principal axis of the anterior naris is in the young child relatively horizontal and projects the air stream backward into the low nasal fossa." This factor is probably responsible for the alteration of the epithelium of the upper surface of the soft palate. This epithelium is ciliated in the fetus but reverts to the stratified squamous type shortly after birth, save for a ciliated epithelium at the extreme posterior margin of the soft palate.

With the growth of the child changes occur throughout the nose and the nasal cavities. Air is directed upward through alteration of the first half inch or more of the nasal cavity, so that little air can pass along the floor of the nose and none enters the meatuses between turbinates. As Proetz [7] pointed out, this constriction must direct the air up and not

5. Johnson, M. R.: Nasal Obstruction and Impairment of Hearing: Forty-Six Cases of Submucous Resection with Audiometric Studies, Arch. Otolaryng. **33**:536 (April) 1941.

6. Proetz,[4] p. 118.

7. Proetz, A. W.: Physiology of the Nose from the Standpoint of the Plastic Surgeon, Arch. Otolaryng. **39**:514 (June) 1944.

backward or pharyngitis sicca and laryngitis sicca are bound to occur. If both are directed backward, anosmia and drying of the eustachian orifice appear.

If these factors assume importance to the plastic surgeon, the drying of the eustachian orifice assumes importance to the tonsillectomist, whether he is a general practitioner, an otolaryngologist or a pediatrician. We have noted a uniformly common finding with all uvulectomized patients—that of pharyngitis sicca.

Delavan [8] stated that the whole posterior surface of the velum is lined with ciliated epithelium which later becomes squamous. We prefer to believe in the remaining barrier of ciliated epithelium, which helps not only to remove small amounts of secretion from the velum but to insure that this secretion passes to a point below the eustachian orifice on the lateral pharyngeal wall.

As Proetz, Hilding [9] and other investigators have long urged, the eustachian orifice must be protected by the surgeon from coming in contact with secretion and from drying if nature has carefully laid out ciliary pathways to avoid these possibilities. The recent phenomenal success of penicillin when used against aural infections in no way nullifies the proper ventilation and inflation of the middle ear in today's and tomorrow's air age. The mucosa about that orifice must remain soft and pliable for effective function of the mechanism opening and closing it.

SELF OBSERVATIONS (FIG. 4)

The observations to be described were conducted quite simply before the mirror and, lest the reader feel that mouth breathing became passé with adenoidectomy, during the common practice of smoking a cigaret. With inhalation of smoke, the air is admittedly dry and hot. Exactly what happens to it below the back of the tongue is a matter of conjecture, particularly if the mouth is closed. The observer had been tonsillectomized successfully, with all structures approximating the normal. An ordinary pen flashlight was used in examining the cavity of the mouth during the observations.

Erect man may breathe through the nose with mouth widely open and velum and uvula relaxed and lowered. No appreciable amount of air enters or leaves the hypopharynx through the mouth. Yet, if a minimal inspiration is used to fill the mouth with smoke and the remaining inspiration passes through the nose with the velum relaxed and lowered, one notes two things:

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8. Delavan, D. B.: Uvula and Soft Palate: Anatomy, in Buck, A. H.: Reference Handbook of the Medical Sciences, ed. 4, revised and edited by T. L. Stedman, New York, William Wood & Company, 1923, vol. 8, p. 399.

9. Hilding, A.: Influence of Ciliary Activity on the Bacteriology of the Nose, Proc. Staff Meet., Mayo Clin. **6**:320 (May 27) 1931.

First there are very slight leakage and interchange between the air of the open but posteriorly closed mouth and the air descending behind the velum. This is shown by visible movement in slight eddies of the smoke. This feature does not alter appreciably with expiration. Second, some of the smoke leaves the mouth to join the air inspired through the nose; the movement of smoke is greater with the lower lip relaxed but is present even though the lips are drawn firmly against the teeth. This feature is exaggerated but evidenced in the cigaret smoker who takes a mouthful of smoke and then draws it through the nose simply by thrusting the mandible and tongue forward with lips relaxed.

To determine the approximate path of air inspired through the mouth, a mouthful of smoke was observed as it passed backward with inspiration

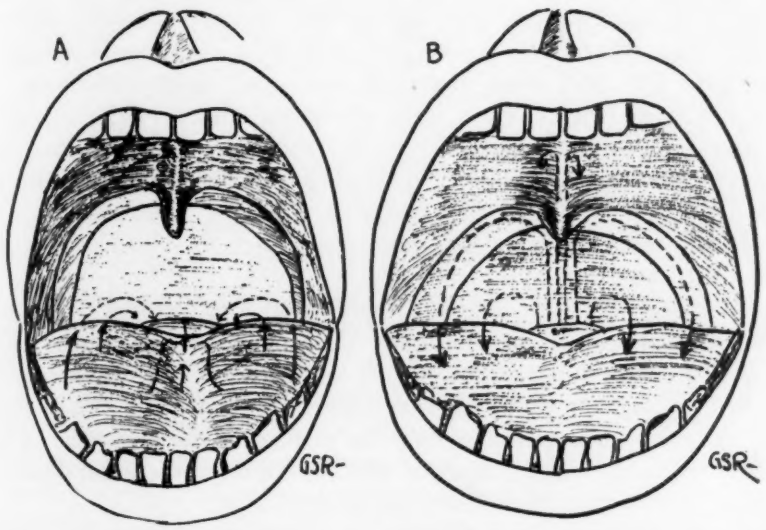

Fig. 4.—*A*, approximate pathways of smoke inhaled through the mouth. Note the deep central furrow of the tongue and the prominence of the longitudinal muscle bundles and of the sulcus terminalis area. The epiglottis is represented by the dotted line. The uvula and the palate are raised, but neither is particularly tense. The drawing represents the mouth of a tonsillectomized person.

B, approximate pathway of exhaled smoke. The uvula and the velum are high and tense. The beam of a pen flashlight interrupting exhaled smoke particles was used to estimate the relative densities of expiratory currents. The drawing represents the mouth of a person who had been tonsillectomized.

completed orally. The velum and uvula ascend; the terminal sulcus and the longitudinal muscle bundles of the tongue become prominent to deepen somewhat the midline raphe of the tongue. The smoke passes backward as shown in figure 4. The smoke disappears first centrally, while currents follow over the longitudinal muscle bundles of the tongue, conforming to the curvature of the teeth and the cheeks. These peripheral currents mixed with the main current at the back of the tongue but were last to disappear.

On being exhaled through the mouth, smoke is altered by its entry into the alveoli, and some mixes with the residual air. The smoke currents consequently cannot be as easily appreciated. However, the beam of the flashlight may be used to determine the relative intensity of air-smoke by the interruption of the beam of light by the smoke particles. As shown in figure 4 *A*, the velum and the uvula again ascend to effect a fair closure of the nasopharynx and oropharynx. Lingual changes are not as apparent as on inspiration. The whitest beam of light appears in the midline, opposite the uvula and over the vault of mouth and palate. More light reaches the periphery of the mouth, showing that expired air travels this pathway as well.

That the uvula is directly affected by an air blast is shown by the flutter action of the structure in a forced expiration without voice. Here it moves anteriorly and can be seen through the open mouth. Emit a high-pitched tone and the soft palate and uvula ascend posterosuperiorly with little if any flutter of the uvula. Subject the uvula to repeated sharp blasts of air from below and the fluttering becomes audible. This mechanism is used as heretofore described, and the structure participates integrally in all whose vocations compel an excessive use of voice. That this flutter is traumatic is evidenced by a frequently irritated uvula and pharyngeal arch in absence of other noticeable cause.

To appreciate all the associated movements that occur in this region with talking, one may hook an index finger over the back of the tongue or pass a soft probe, curved to fit the tongue, backward during speech.

The importance of the normal palate, the uvula and the anterior and posterior pillars in protecting the orifices of the eustachian tubes cannot be underestimated, and the effects of altering the normal pattern through operative procedure will be outlined later.

THE LITERATURE

Ewens,[10] who attributed many of the ills of man's upper respiratory tract to the uvula, advanced the theory that this structure is a mere vestigial remnant of an upward regressing velum found in lower animals; that man, no longer having need of a protective curtain in the throat to prevent the ingress of insects and other foreign objects occasioned by his running through the forest primeval panting, has, over the centuries, lost the original character of the palate. The uvula is, according to this author, the vestigial remnant.

Articles[11] have appeared in the literature describing both partial and total resection of the uvula in Italian East Africa, Arabia, Kordofan

10. Ewens, A. E.: An Overlooked Factor in Susceptibility to the Common Cold, Virginia M. Monthly **61**:40 (April) 1934.

11. d'Arcangelo, D.: Partial Uvulectomy in People of Italian East Africa, Boll. Soc. ital. di med. e ig. trop. (no. 3) **4**:445, 1944. Sarnelli, T.: Resection of Uvula in Native Medicine of Arabia, Kordofan and Ethiopia, Riv. med. trop. **4**: 288 (July-Sept.) 1940.

and Ethiopia. Although these articles were not available for study, it is reasonable to expect a primitive people to excise the uvula. Their probable reasoning, in the absence of medical care as we know it, is logical.

If the tonsils exhibit chronic hypertrophy from some cause and the uvula is edematous to the extent of hindering respiration or eating, the latter structure is going to be excised. It is the only one easily grasped and removed in the reestablishment of some degree of normal function within the mouth.

Delavan [8] stated that the uvula acts as a drain for the mucous secretions from the nasal cavities, directing their flow toward the base of the tongue, whence they are expelled. This view is certainly tenable when one witnesses the mucous strands which are often seen extending from the uvula to the back of the tongue. We also feel that the bulk of a tenacious secretion in the midline may be transferred to the posterior pharyngeal wall by the uvula, and, if expectoration is possible, the uvula and the soft palate may ascend ahead of the tongue to establish contact with the secretion on the posterior pharyngeal wall. As the tongue is lowered, the uvula can then transfer the secretion back to the tongue for expectoration. "Hawking" and expectoration consume only a few seconds at most.

Although the uvula is in the midline above and the valleculae, the epiglottis and the larynx are below, it is not unreasonable to assume that they are more intimately connected with the structures of the mouth and hypopharynx as regards collection of and protection from the secretions therein.

Steinberg [12] has reported edema of the structure following the administration of meperidine hydrochloride ("demerol hydrochloride") and scopolamine hydrobromide to produce obstetric analgesia. De Juan [13] reported the submucosal hemorrhage of the uvula that suddenly appears without evident cause and stated that the phenomenon is similar to subconjunctival hemorrhage—the cause of which is likewise unknown. Thomsen [14] stated that he had observed 2 or 3 cases and had heard of others in which there was no suspicion that the uvula had been traumatized in any way during operation as it was seen to be intact and its function unimpaired before the patient left the table; yet, within ten to twenty

12. Steinberg, M.: Edema of Uvula and Edema of Glottis—Reaction to Demerol-Scopolamine Analgesia, Am. J. Obst. & Gynec. **50**:542 (Nov.) 1945.

13. de Juan, P.: Apoplexy of the Uvula: Three Cases, Rev. clín. españ. **8**: 268 (Feb. 28) 1943.

14. Thomsen, St. C., and Negus, V. E.: Diseases of the Nose and Throat: A Textbook for Students and Practitioners, ed. 4, New York, D. Appleton-Century Company, Inc., 1937, p. 418.

days the uvula had, without any appearance of bruise, traumatism, swelling or inflammation, quietly shrunk and atrophied to a small, wasted prominence or entirely disappeared. In 1 case there was a slight cleft palate type of speech for a time, but this disappeared. The uvula is, without doubt, subject to, and participates early in, scarcely otherwise discernible vascular change of the mucous membrane from whatever cause.

Ewens [15] stated "that in spite of wide publication at home and abroad for the past twenty-five years staphylectomy (uvulectomy) is not widely accepted." He expressed the opinion that complete excision of the uvula will accomplish free nasal ventilation almost without exception within seventy-two hours. Further, diminution of nasal secretion and of post-nasal drip, with cessation of early morning paroxysms of "hawking," occurs, with an unquestioned reduction in susceptibility to colds. There is also prompt symptomatic relief from painful involvement of sinuses (frontal and maxillary) which is truly dramatic in the majority of cases. He had performed staphylectomy in 3,000 cases and reported 1 per cent of these cases which "symbolize the salient remedial features of staphylectomy," but he awaited an equal number of histories from unstaphylectomized controls for full establishment of his theories.

It has not been our good fortune to see these symptoms vanish in those who have been inadvertently uvulectomized in tonsillectomy. All uvulectomized patients exhibit to a greater or less degree pharyngitis sicca. We hope that the indiscriminate removal of the uvula will not be widely accepted other than in Arabia, Kordofan and Ethiopia and for the reason previously mentioned.

CASES

The cases to be mentioned were, with the single exception of the first case, collected in a single clinic week at the Manhattan Eye, Ear and Throat Hospital. Similar cases could be multiplied many times and seen in almost any city or town in the United States.

1. Cases demonstrating that the uvula is intimately associated with nasopharyngeal health and arguing for preservation of all structures in tonsillectomy:

(*a*) H. A., a 45 year old pilot with 18,000 hours flying time and eighteen years of experience with air transportation discovered that acuity of hearing was diminished for high (radio) tones five years after tonsillectomy. An audiogram (Maico) confirmed this, showing a dip at 4096 double vibrations, worse on the right, with recovery of higher tones in both ears. The symptom most disturbing to the patient was a "popping" in both ears, worse on the right. Examination

15. Ewens, A. E.: Staphylectomy for Control of Colds and Sinusitis (Epochal Anticatarrhal Innovation), M. Press **210**:251 (Oct. 20) 1943.

revealed that the tonsillectomy had been poorly performed in that the right posterior pillar was little more than a strand of muscle, with fibrous contracture and right deviation of the uvula. There was a noticeable reduction of hearing for the spoken voice, although in the same audiometric range the reduction was not apparent.

Comment: The audiometric dip is not significant in view of the patient's long association with motor noise. "Popping" has been described as a little understood, frequently occurring and ofttimes disturbing phenomenon. It may be due to alteration of the normal eustachian tube pressures, possible drying of the orifice and alteration of the action of the right tensor veli palatini muscle, with consequent change in the function of the orifice.

(*b*) M. B., a white woman aged 32, entered the clinic, complaining of pain in the throat, pain in the left ear and a foul taste similar to that of sour cream, with a reduced appreciation for tasty foods. These symptoms followed tonsillectomy, performed three months previously, with a stripping away of the tongue from the tonsillar fossa later. Examination revealed a shortened uvula deviating to the left, with a straightened pharyngeal arch on the left, the straightening being due to fibrous contracture.

(*c*) O. W., a Negro woman aged 31, complained of intermittent tinnitus and pain in the left ear following tonsillectomy, performed five years previously. Examination revealed absence of the anterior pillar and the tonsil on the left, with the uvula shortened and deviating to the left. The pharyngeal arch was straightened, and pharyngitis sicca was evident.

Comment: These 3 cases, multiplied many times, indicate that a better realization of the importance of the pharyngeal structures is needed today or until such time at least as other means than nature's own eustachian tube and orifice for the inflation of the highly important middle ear may be developed. Needless to say, care must be exercised during tonsillectomy.

2. A case indicating that the uvula may be a midline reenforcement of the soft palate in voice control:

R. S., a white woman aged 25 who had been singing since the age of 6, entered the hospital because of an ear complaint. At the age of 10 she had undergone adenoidectomy and tonsillectomy. Whereas, according to her, she had previously been a lyric soprano, she has never gone above the range of contralto since her operation. She had further voice training and much practice. She admitted that "her voice might have changed about the age of 14 but she could not sing as long as previously." Examination revealed the uvula was absent and that there was a straight line of fibrous tissue for palate. The throat was dry. There were no prominent symptoms of disease of the ears.

Comment: The uvula must have such a function, that of reenforcement, and hence that of prevention of nasal quality of voice or "rhinolalia aperta," noted by Proetz, Thomsen and many others as following the operative removal of the uvula. The greatest force of the air column, in the production of high-pitched tone, is directly over the larynx. A straight band of fibrous tissue may alter the resonance factor. However right or wrong in her deduction, the patient attributed her singing difficulties to a poor performance of adenoidectomy and tonsillectomy. She lost the opportunity of becoming a great lyric soprano, and with the legal consciousness of patients today, one may expect an increase in medicolegal inquiry with regard to this problem.

3. Cases in which the uvula served as the warning flag of the pharynx:

(*a*) W. K., a 46 year old Negro, entered the emergency room complaining of soreness of the throat of three days' duration. There was an old history of repeated peritonsillar abscess without tonsillectomy. Examination revealed an enlarged, edematous and reddened uvula. Indirect examination of the larynx, although the patient evidenced no respiratory difficulty, revealed edema of the valleculae and of the posterior surface of the epiglottis, with the edema beginning to extend along both aryepiglottic folds. The patient was advised to enter the hospital for chemotherapy and refused.

(*b*) W. F. D., a 24 year old Negro man, entered the clinic, complaining of respiratory difficulty. Thirty-six hours previously soreness and dryness of the throat had developed, rapidly progressing to obstructive laryngeal dyspnea. There was a past history of repeated attacks of tonsillitis without tonsillectomy. Examination revealed a huge edematous and elongated uvula. Direct laryngoscopy revealed edema of the epiglottis and the aryepiglottic folds sufficient to occlude the rima vestibuli. Tracheotomy was performed, and the patient was discharged on the thirteenth hospital day with the tracheotomy wound closed and the edema completely reduced.

Comment: That the uvula participates in scarcely perceptible vascular and lymphatic changes occurring in the pharynx has been long known and cited heretofore. That the structure is a visible means of ascertaining possible changes occurring below the base of the tongue, with the institution of possible life-saving measures, is shown.

4. No experimental means has been devised to show that the uvula, in its massaging of the posterior pharyngeal wall, performs a useful function in clearing the base of the adenoid in children. Secretions are removed from a constantly diseased focus of infection of the middle ear.

It is reasonable to assume that the rate at which infected material drains from the adenoidal folds is heightened if the secretion moves downward from the base on a layer of squamous epithelium.

That this is an inefficient system at best is shown by the larger number of middle ear infections in nonadenoidectomized children. Repeated infection of the ears is an indication for adenoidectomy. Anatomists have described the mucosa of the adenoid as being thrown into transverse folds. In our experience the most constant transverse fold is that found at the base and is formed by the superior constrictor muscle of the pharynx.

Our observations in the operating room would lead us to believe that the mucosa is thrown more often into ridges running to a central vertical furrow or into a series of overlapping V's, pointing toward the hypopharynx. If the surface of the adenoid is traumatized to cause oozing, in the recumbent patient the blood drains toward the oropharyngeal aspect of the adenoid.

A situation analogous to that of the functioning of the uvula in such cases exists in the way any thick, viscid material moves down a perpen-

dicular or inclined plane if it is applied above a transverse line. For a while the whole line advances, until the cohesive forces and the surface tension are interrupted at one point, at which time the flow is directed at that point. For instance, if a finger is drawn through the line and moved downward, the flow moves through the point of interruption of surface tension. It is easily seen that in adenoidal infections in children it is better to have the interruption of flow extend down the posterior pharyngeal wall equidistant from the two eustachian orifices.

WHEN SHOULD THE UVULA BE SUBJECTED TO OPERATIVE PROCEDURE?

Lederer and many others have stated that the uvula should not be excised or incised unless it is afflicted with a cancerous change or is producing annoying or distressing symptoms of redundancy. The uvula must be freed of hanging shreds of mucosa after tonsillectomy. The following cases are cited as evidence that the forest is still being missed because of the trees and that these old truths are being forgotten.

(*a*) A Negro man aged 45 presented himself at the clinic with a complaint of chronic cough. Three years previously he had undergone tonsillectomy. He now had a sensation of "something in his throat." Examination revealed a long, redundant bit of mucosa affixed to the posterior surface of the uvula, which probably had come from a posterior pillar during the operation. It irritated the pharynx and the epiglottis, producing a sensation of "something in the throat" and a chronic cough—both of which ceased with removal of the mucosal shred.

(*b*) I. S., a 58 year old white man, entered the clinic, complaining of a chronic cough which he had had for more than one year. He described it as an aggravating nervous cough due to a sensation of "something in the throat." He had seen his family physician and had undergone numerous medical and roentgenologic examinations which disclosed no evidence of disease. Examination revealed a large, redundant uvula about 1½ inches (about 4 cm.) in length and extending below the base of the tongue with velum up. When almost ½ to 1 inch of this uvula was removed, there was an immediate cessation of the symptoms that had caused this patient much discomfort and loss of time in medical examination.

FUNCTION OF UVULA SUMMARIZED

1. It massages and moistens the cellular structure of the posterior pharyngeal wall.

2. It aids in the removing of material from the posterior pharyngeal wall and in the passing of this material downward to the hypopharynx and the base of the tongue.

3. It serves as a transfer organ to bridge the deficiency between the velum and the posterior pharyngeal wall as secretion moves from the midline above and anteriorly to the midline posteriorly.

4. It is a valuable aid in preventing middle ear disease in children.

5. By participating in the preservation of normal pharyngeal arches the uvula prevents pharyngitis sicca with subsequent drying of the

eustachian orifices as might be expected with a high arch on one side or with a fibrous straight line contracture.

6. It is the warning flag of the pharynx in many instances.

7. It prevents rhinolalia aperta in successful tonsillectomy and is a valuable adjunct to voice control, possibly as a midline buttress, particularly in singers. This function is of little value in the speaking ranges apart from preventing unpleasant qualities.

8. A uvula deviating to one or the other side may indicate a weakness of the accessory nerve on the opposite side.

CONCLUSIONS

The uvula is a valuable means of preserving and detecting nasopharyngeal health and change. It should not be traumatized needlessly, nor should it be subjected to operation unless a definite symptom complex traceable to it is evidenced.

More must be known about the eustachian tube and its nasopharyngeal orifice before one becomes willing to sacrifice a uvula or to class it as a vestigial remnant. To ignore an edematous uvula is as negligent as to ignore chronic hoarseness without ascertaining the state of the vocal cords by viewing the anterior commissure in indirect laryngoscopy.

210 East Sixty-Fourth Street (Dr. Richardson).

30 East Fortieth Street (Dr. Pullen).

COMBINED SEPTAL RESECTION AND RHINOPLASTY FOR RESTORATION OF NASAL RESPIRATORY FUNCTION

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THIS ARTICLE concerns itself entirely with indications and operative procedures for the restoration of the respiratory function of the nose. In accomplishing this thoroughly, it is often necessary for the rhinologist to employ some of the rhinoplastic procedures. It may be argued that the rhinologist can perform the septal resection and have the plastic surgeon do the rest. I have found this impracticable. As evidence of the impracticability, there is the lack of agreement as to which should be done first. Byars [1] stated that the rhinoplasty should be done first, as too much septal resection may interfere with the plastic work later. On the other hand, Eisenstodt [2] pointed out that lateral osteotomy narrows the nasal chamber. If there is already an obstructive deviation, it may not be possible to perform osteotomy.

It has been my experience that the standard submucous resection fails in a great many cases because the resection of the septum alone is not sufficient. I have found that in only 1 of every 3 cases of nasal obstruction due to deviation of the septum can the condition be corrected by simple submucous resection. In the remaining cases there are other deformities which must be considered. The correction of these other deformities falls into the realm of plastic surgery, but the plastic procedure required is of a type which the rhinologist should be able to carry out.

I have devised an operation which combines several established technics with modifications. For each case two or more of the technics are combined to meet the specific requirements.

OPERATION

1. *Resection of the Septum.*—This is especially suitable when there is a subluxation of the anterior portion of the septal cartilage, but is applicable for any

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1. Byars, L. T.: Surgical Correction of Nasal Deformities, Surg., Gynec. & Obst. **84**:65 (Jan.) 1947.

2. Eisenstodt, W.: Management of Septal Deformities, Arch. Otolaryng. **45**:77 (Jan.) 1947.

deviation. The columella is pushed aside so that the anterior part of the septum is exposed. A vertical incision is made over the cartilage. The mucosa and perichondrium are now freed over the concave side only. The separation is carried down to the floor and along the floor if necessary. The next step is to incise the cartilage and attached mucoperichondrium along the floor for about 2 or 3 cm. An incision is now made through the cartilage about 2 cm. from its front

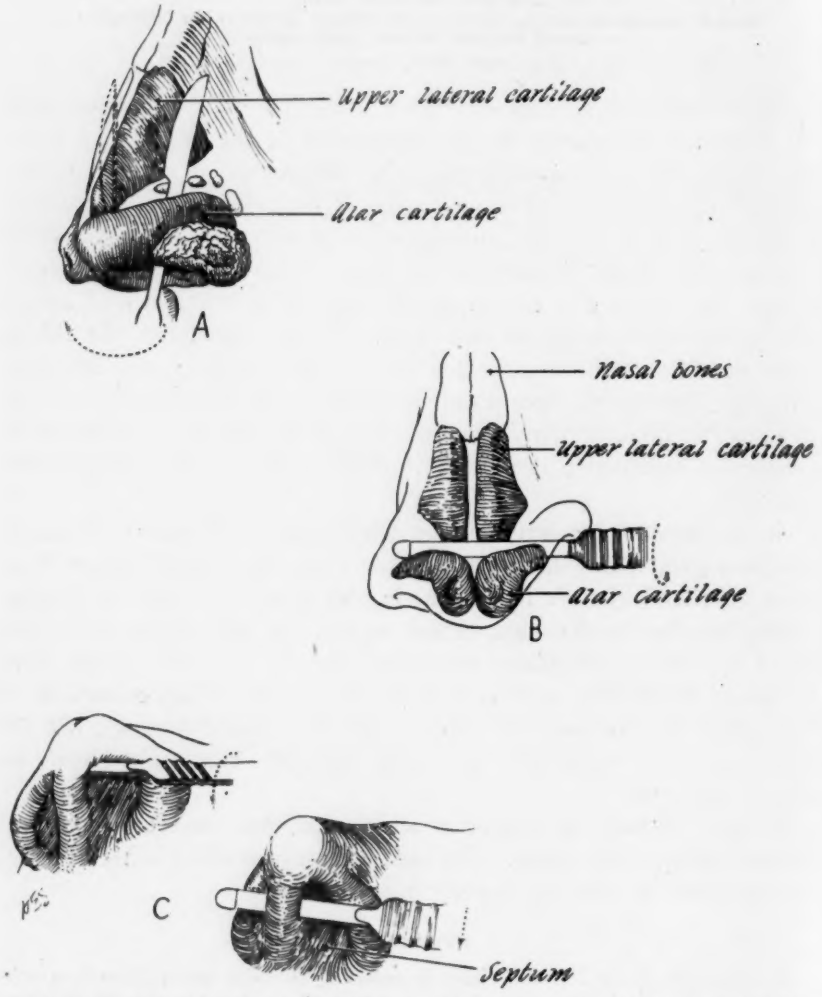

Fig. 1.—Basic rhinoplastic preparatory procedure: *A*, carrying the incision up over the lateral cartilage and the nasal bone and freeing the soft tissues over the bridge of the nose. This is done on both sides of the nose.

B, connecting the right and left pockets and extending the incision to the tips of the lateral cartilages.

C, bringing the incision down between the columella and the septum.

end. The attached mucoperichondrium is elevated, and the deviated septum resected by the usual technic. The remaining anterior portion is now placed in

the midline. If it does not fall there readily, one is to determine what is holding it. Since it is free on the floor, the point of resistance may be its attachment to one or the other of the upper lateral cartilages, or the cause may be a bowing of its horizontal part. The upper lateral cartilages can be severed from the septum, and a bowing can be overcome by taking a wedge out on the convex side and fracturing the septum over.[3]

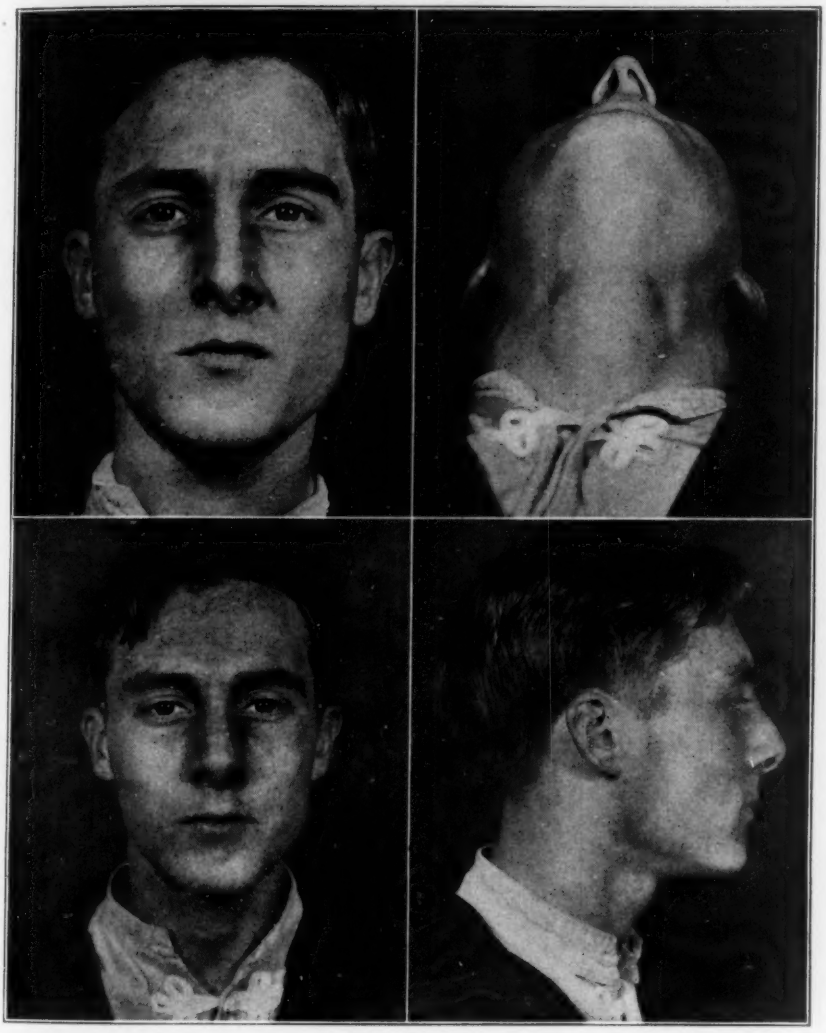

Fig. 2.—The upper photographs show a patient with dislocation of the nasal septum and lateral displacement of the bridge; the lower ones, the correction obtained by operation.

2. *The Basic Rhinoplastic Preparatory Procedure* (fig. 1).—This procedure is the standard approach to any intranasal rhinoplastic correction. An incision is

3. Metzenbaum, M.: Replacement of Lower End of Displaced Septal Cartilage, Arch. Otolaryng. **9**:282 (March) 1929.

made between the upper lateral and the lower alar cartilage. The incision is carried upward over the upper cartilage and the nasal bone, freeing the soft tissues over the bridge of the nose. A periosteal elevator may be used instead of the scalpel, although I prefer the latter. This is done on both sides, and the pocket created on one side is joined to that on the other. A knife with a protected point

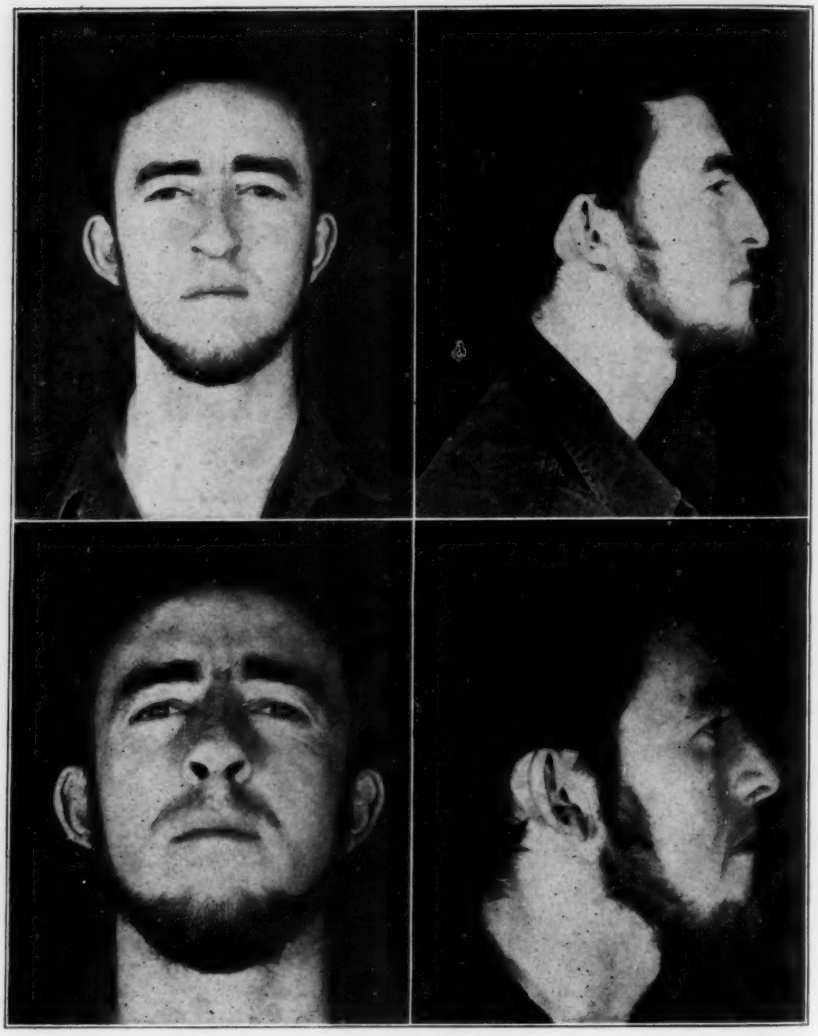

Fig. 3.—Hump deformity of the nose. The upper photographs show the preoperative, and the lower ones the postoperative, appearance of the nose.

is now inserted through one incision, turned at right angles, and the end pushed out through the other with the cutting edge facing the operator. The knife is now drawn forward to the tips of the upper lateral cartilages and over the end of the septum, and then downward between the columella and the septum.

3. *Correction of Deformities of the Nasal Bones.*—(*a*) Hump: If this is present, it is removed with a bayonet saw and smoothed out with a rasp. Instead of the bayonet saw I often employ a narrow rongeur, the upper edges of which have been cut away. A dental bone-trimming forceps, angulated, is often used near the glabella. Kazanjian[4] has recently designed a rongeur for this purpose.

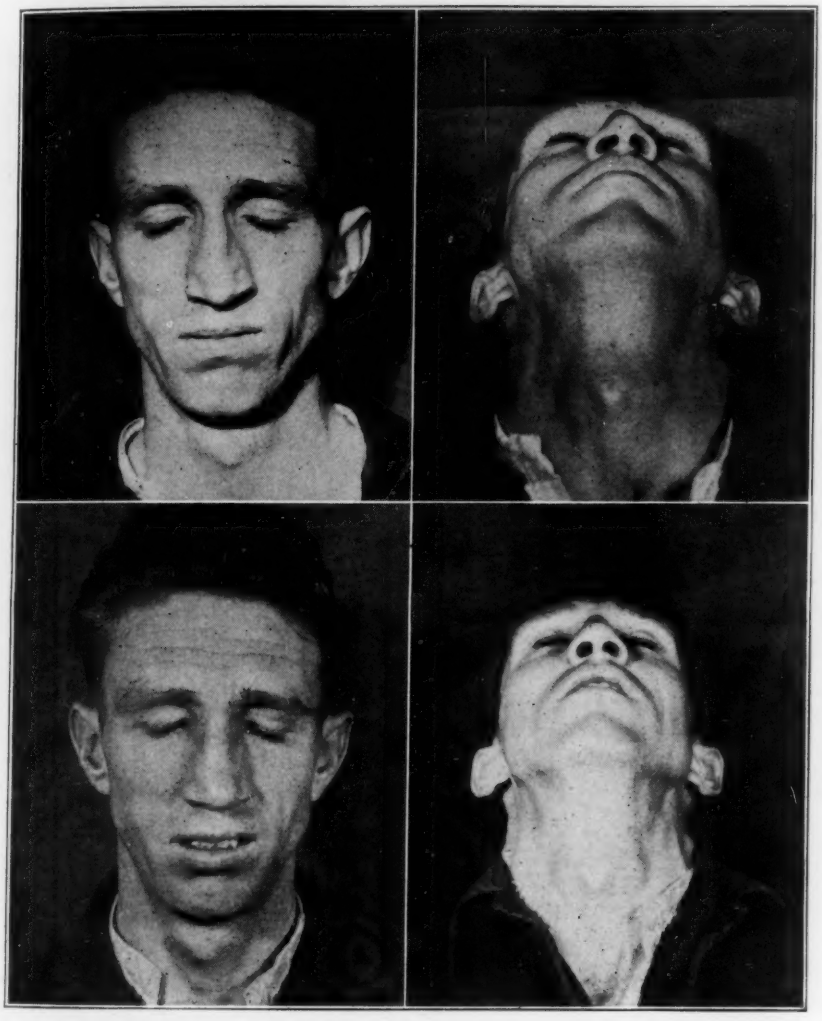

Fig. 4.—Hump deformity of the nose, with sagging tip. The lower photographs show the nose corrected by operation.

(*b*) Lateral Deviation: Saw cuts are made at the base of the concavity and the convexity and the entire vault is infracted into position. It may be necessary to introduce an Asch forceps, one blade over the nasal bone, the other into the nose, and thus loosen the nasal bones.

4. Kazanjian, V. H., and Holmes, E. M.: New Rongeur for Removal of Nasal Hump, Arch. Otolaryng. **45**:361 (May) 1947.

With a lateral deformity of the nasal bones, one or both of the frontomaxillary processes are usually involved, an osteotomy of these structures may be necessary.

4. *Correction of Deformities of the Frontomaxillary Processes (Lateral Osteotomy):* Here the original basic rhinoplastic preparatory procedure is inadequate. Another incision has to be made in the piriform space, and the soft tissues over the lateral aspect of the nose have to be elevated so that a Joseph's saw can be

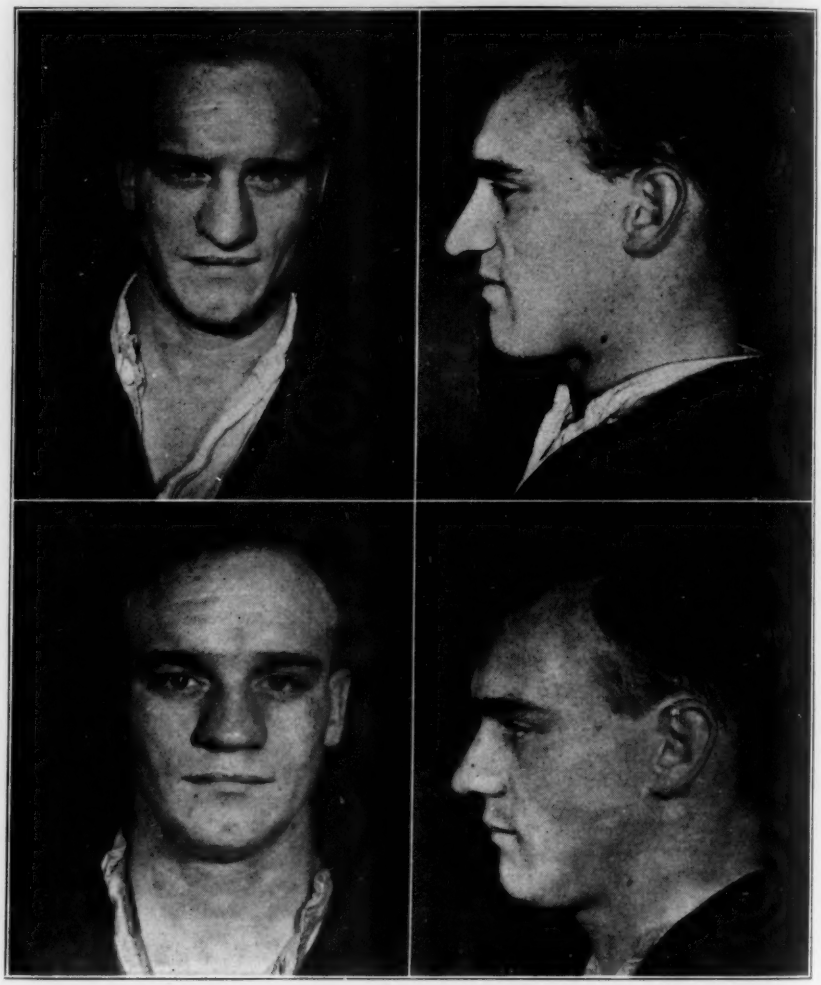

Fig. 5.—Hook nose with sagging tip. The upper photographs show the preoperative, the lower ones the postoperative, appearance.

introduced. The frontomaxillary process is sawed through and then infracted medially or laterally as the case requires. The use of the Asch forceps, by putting one blade over the bone and the other in the nose, can be applied here as in the case of corrections of the nasal bones.

5. *Correction of a Depressed Nasal Tip.*—This is easily accomplished by removing a triangular part of the septum at its anterior extremity with the base up.

After the ends of the upper lateral cartilages have been cut away from the septum with nasal scissors, the septum is seized with a mouse tooth forceps and brought out of one of the nostrils. The triangle is excised with a scalpel or scissors, and the lateral cartilages are shortened proportionately.[5]

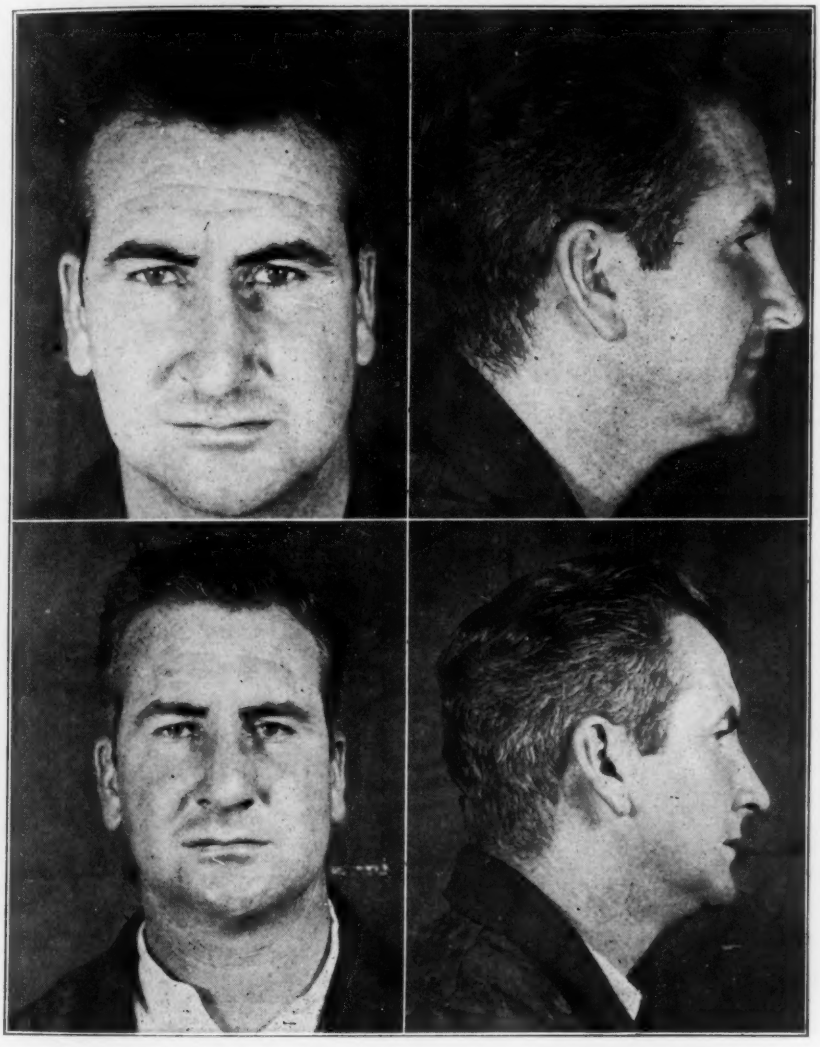

Fig. 6.—Hook nose deformity. The preoperative photographs are shown above; the postoperative ones, below.

6. *Correction of the Upper Lateral Cartilages.*—In long-standing deformities the size of the two cartilages may be different; one may be atrophic and the other hypertrophic. The large one can easily be reduced by excising a part of it.

5. Aufricht, G. A.: A Few Hints in Surgical Details in Rhinoplasty, Laryngoscope **53**:317 (May) 1943.

Where there is extreme atrophy a piece of excised septal cartilage can be inserted into the pocket.

7. Correction of the Lower Alar Cartilages.—These may be reduced in size by resecting a rim from the posterior extremity. They may be advanced or recessed, whichever is necessary, by sewing them to the septum with appropriately placed sutures. A section of the vertical strut can be excised if the height is too great.

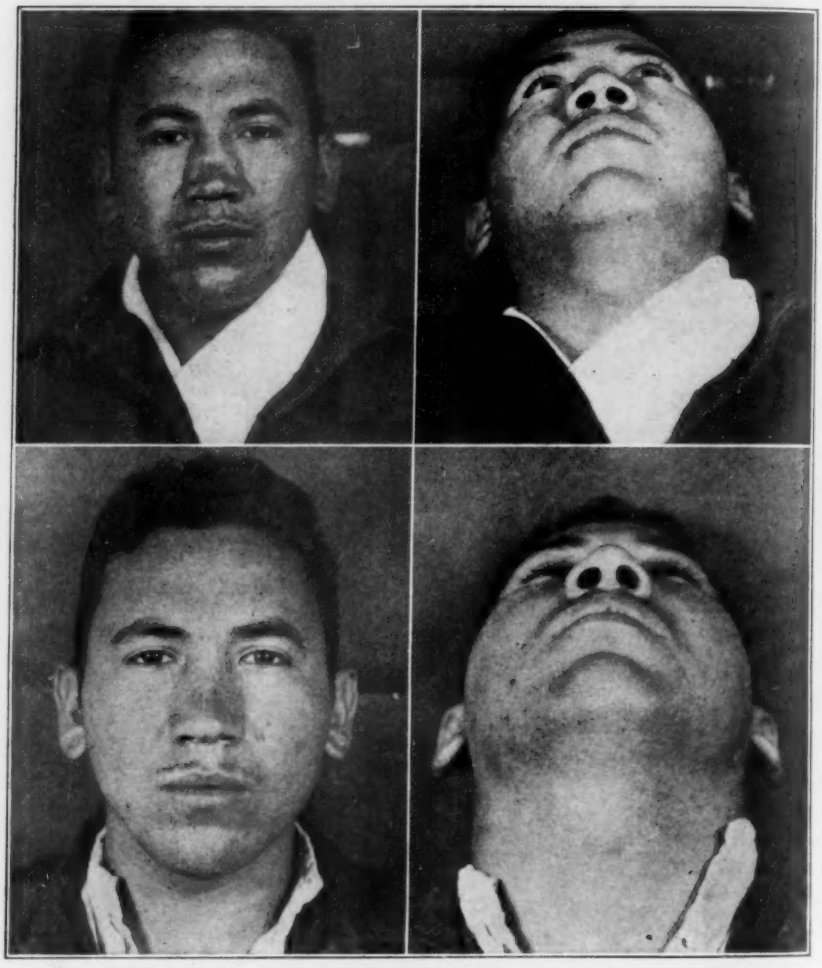

Fig. 7.—Saddle nose deformity with depressed upper lateral cartilages. The upper photographs show the preoperative appearance; the lower ones show the nose corrected with a cartilage graft, a columellar wedge and a "gable roof" anchor at the upper lateral cartilages.

The procedures described will meet most of the requirements for restoring functions and correcting deformities. The individual case requires a blending of these procedures.

There is another group of deformities and functional disturbances which may require grafts. The saddle nose, if due to loss of septum,

either surgical or infectious, seldom requires a submucous resection. I have had great success with preserved cartilage grafts.

The question whether to put in a columellar support or not depends on whether the graft will overlie the nasal bones or not. If it does, it will become aherent

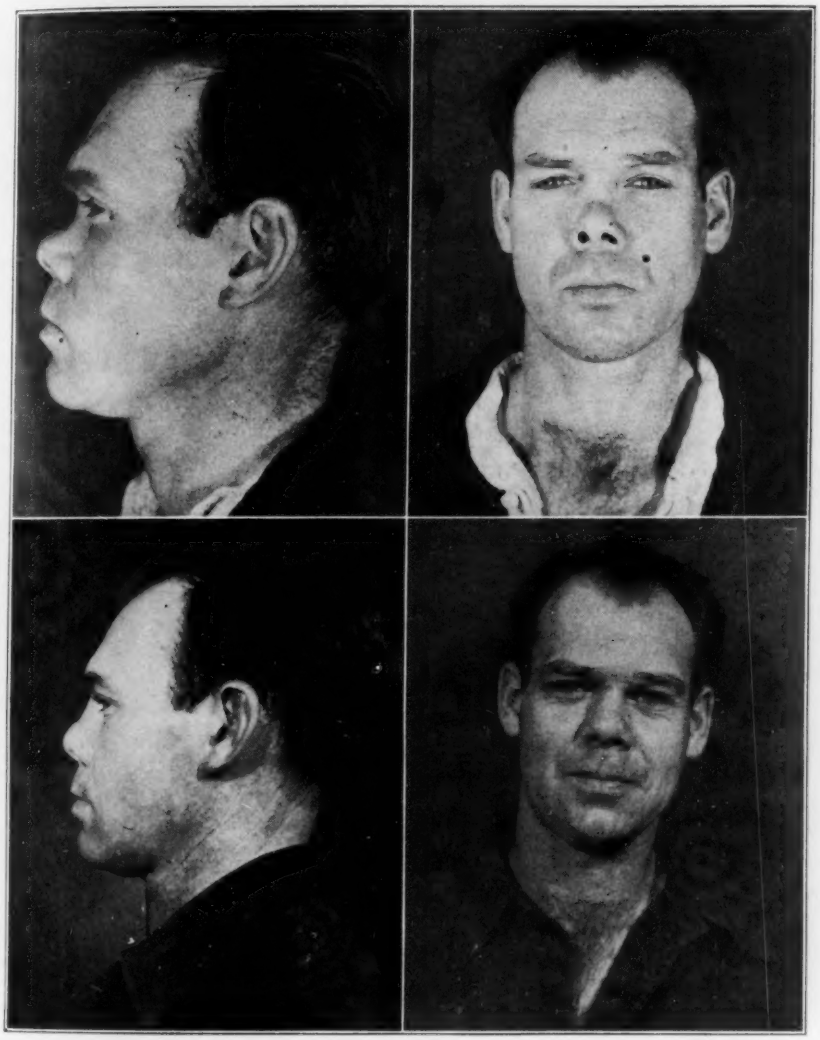

Fig. 8.—Marked saddle nose as it appeared before operation (upper photographs) and after correction with cartilage grafts.

to the periosteum and provide excellent support. If it commences at the ends of the nasal bones, I make a pocket in the anterior part of the membranous septum and insert a cartilage wedge. The upper horizontal strut rests on this. It is quite simple to do this and does not require the carving out of angulated pieces of cartilage. The basic rhinoplastic preparatory procedure is carried out with the

exception that the incision is made only on one side and not brought down between the end of the septum and the columella.

If the upper lateral cartilages have sagged down, they are freed laterally and then anchored above the cartilage graft so that they form a gable roof effect. A mattress suture is placed in the midportion of the cartilage, both ends being brought out somewhat to the opposite side and tied over a dental cotton roll. This causes the upper lateral cartilages to adhere to each other and to the overlying skin. The latter is held up by the cartilage graft.

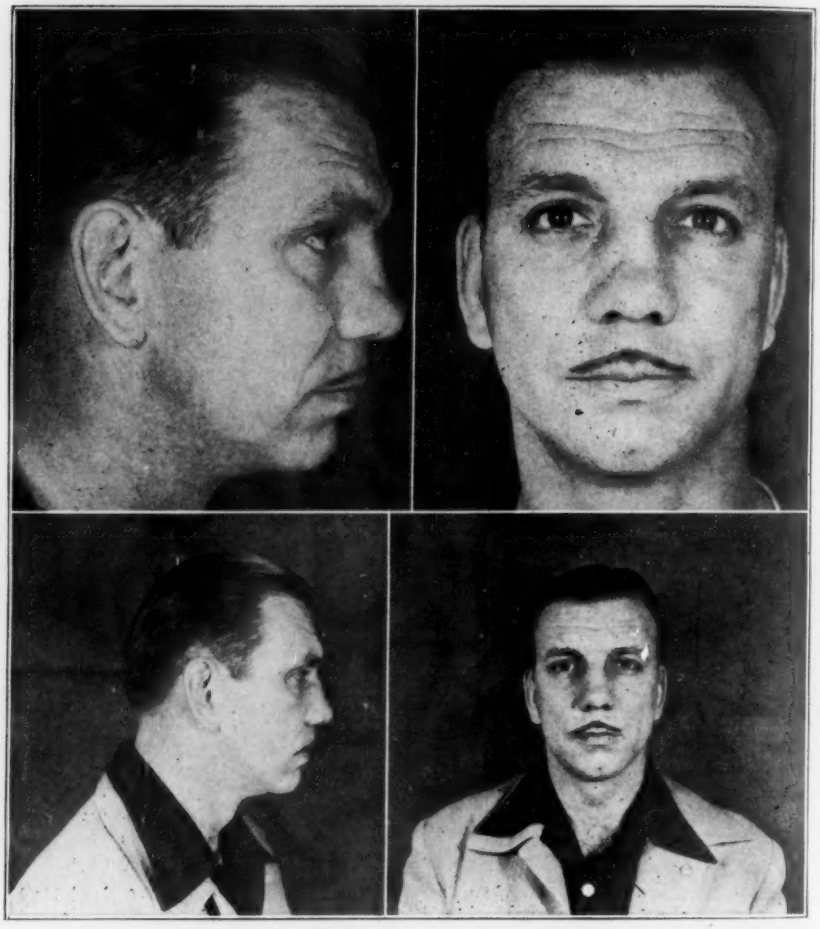

Fig. 9.—Atrophy of the right upper lateral cartilage. The upper photographs show the preoperative appearance of the nose; the lower ones, the nose corrected by insertion of a graft.

Method of Treating Cartilage.—Cartilage is usually obtained at autopsies or at operations for thoracic conditions. All soft tissue is removed, and the cartilage is placed in an aqueous solution of "merthiolate" 1:4,000 for seven days. It is then placed in a fresh solution for another week, after which time it is ready for use. It can be kept indefinitely in a refrigerator. Two days before use the solution is tested bacteriologically.

Anesthetic.—The anesthetic used is a 2 per cent solution of procaine hydrochloride with 8 drops of epinephrine solution U.S.P. to 1 ounce (31 cc.). Cocaine is not necessary. The anesthetic solution is infiltrated submucosally. An aseptic technic is followed.

Closing Incisions.—The incision between the columella and the septum is sutured with one or two triple O silk sutures. They should be carefully placed so as not to disturb the nasolabial angle. The incisions between the upper lateral and lower alar cartilages need not be closed except when a graft has been inserted; then careful closing is necessary. The incisions for lateral osteotomy need not be closed.

Packing.—Strips of gauze saturated with penicillin jelly, 1,000 units per cubic centimeter, are used.

Splinting.—Adhesive tape is used for the first twenty-four hours. After that a thin aluminum or copper splint is molded and encased in adhesive tape. This is made to adhere to the original adhesive tape with collodion. The splint is kept on for about eight or ten days.

Postoperative Medication.—The patients are given 25,000 units of penicillin every three hours for at least forty-eight hours postoperatively.

TYPES OF DEFORMITIES AND APPLICATION OF THE PROCEDURES DESCRIBED

All of the patients came for consultation primarily because of nasal obstruction and were operated on for this reason. Cosmetic results were incidental. Resection of the septum and the basic rhinoplastic preparatory procedure were carried out in all of them.

Hook Nose.—This is a common deformity. In addition to the two basic procedures, correction of lateral deviation of the nasal bones, reduction of the hump, mesial or lateral osteotomy and elevation of the tip may be necessary.

Hump Nose with Depressed Tip.—Here the two basic procedures and sawing off of the hump, closing of the gap by osteotomies with mesial infraction of the frontomaxillary processes, and elevation of the tip are necessary.

Saddle Nose.—Resection of the septum may or may not be necessary. A limited basic rhinoplastic preparatory procedure is carried out with only one vestibular incision between the lateral and alar cartilages. A prepared graft is inserted and a wedge placed in a pocket in the membranous septum if anterior support is necessary. If the lateral cartilages sag, they are thoroughly mobilized and anchored to the soft tissues overlying the cartilage graft.

The accompanying photographs show the results obtained on some of the patients. As stated, the primary aim was for function; appearance was considered only secondarily.

SUMMARY

The submucous resection alone is inadequate in about two thirds of the cases of nasal obstruction. Rhinoplastic correction of deformities of the nasal bones and the other structures of the external nose becomes necessary. Both procedures can be carried out in one operation. The septal resection and the rhinoplastic procedures are outlined.

RHINOPLASTY

The Use of Methyl Methacrylate Implants

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AND
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THE VARIOUS materials used in the reconstruction of depression defects of the nose are almost as numerous as the decades since rhinoplasties were first mentioned in writings, about the seventh century B.C.[1] Surgical implants have varied from paraffin to bone, and none has been found to be entirely satisfactory.

In 1942 one of the authors (R. S. L.) while at the Mayo Clinic[2] noted the successful use of methyl methacrylate, an acrylic resin, in the reconstruction of a hip joint. This resin, commonly known as "lucite," was then considered in the ear, nose and throat department of the United States Marine Hospital, Baltimore, for use as nasal implants. This type of surgical implantation was first done at this clinic in January 1943, and since then 20 patients have received surgical implants.

Because it was feared that the tissue might react to methyl methacrylate as to a foreign body, the material was implanted first in the subcutaneous tissue of the abdominal wall of a dog and observed for one month. When it proved inactive at this site it was implanted in the stump of a pedicle graft which had already been used on a human face. The methyl methacrylate again was found to be inert, as proved by microscopic sections of the pedicle one month later. Synthetic implants of this type have been used by us and by others for facial deformities. Brown[3] reported their use and gave a good description of their properties. We feel that their greatest advantage over other types of implants now used

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From the United States Marine Hospital.

* Formerly Surgeon, United Stated Public Health Service.

1. Hirschberg, J., cited by Hughes, W. L.: Reconstructive Surgery of the Eyelids, St. Louis, C. V. Mosby Company, 1943, p. 20.

2. The method was developed by Dr. Paul H. Harmon of the Robert Parker Hospital, Sayre, Pa., using "lucite" (Plastic Cups for Arthritic Hip Joints, abstracted, Clin. Med. & Surg. **49**:25 [Jan.] 1942).

3. Brown, A. M.: Sculpturally Molded Synthetic Implants in Plastic Surgery, Arch. Otolaryng. **39**:179 (Feb.) 1944.

is the ease of accurate molding and processing. The implant holds its shape, and a secondary operation is avoided, such as is necessary when fresh cartilage is used.

PREPARATION OF THE IMPLANT

The methyl methacrylate[4] which we use is the same as that which can be purchased at the dental supply houses and which is used for the construction of prosthetic dentures. This product as supplied consists of a powder, which is fully polymerized methyl methacrylate, and the monomer, which is liquid. The monomer, the solvent for the polymer, is introduced to render the mass plastic so that it may be pressed into any desired shape. The curing process consists of polymerizing the monomer under pressure and in a hot water bath. The material will be found to be a transparent, relatively stable and fairly strong thermoplastic resin. It causes no tissue reaction when properly processed. The liquid monomer, however, is irritating unless properly tied up with the polymer by adequate heat under pressure. We have found ordinary boiling under pressure as described later to be quite satisfactory.

Originally the size of the splint to be used was determined by making a plaster cast of the patient's face and building up the defect of the nose with wax. However, we have found that it is just as satisfactory, and less involved, to fashion a splint of dental impression wax directly on the patient's nose. Here we determine the thickness, the length and the curvature of the dorsal surface of the necessary implant, plus the approximate length of the base of its L-shaped portion. The wax model is made somewhat heavier than the final product to insure against breaking of the final splint as it is removed from the flask. After the future implant has been properly processed, it is again measured with the patient as a model and ground to the desired size and thickness by means of a dental drill and an emery wheel. It is then perforated with numerous dental burr punctures (size 8 or 9) approximately 1.5 mm. in diameter. The final product is then polished with a buffer. Care is taken not to shorten the base of the "L" excessively, as this may be done at the time of operation with bone rongeurs or with a dental drill. This drill is covered with sterile drapes and kept beside the operating table for making any changes in the splint which may be found necessary.

The technic used for the laboratory construction of the implant is the standard one used for processing dentures. The wax pattern is flasked in dental stone, the splint mold technic being used. The flask is separated, and the wax is flushed out with boiling water. The two halves of the flask can be lined with tin-foil, or they can be coated with a commercial acrylic separating fluid.[5] The acrylic material is then prepared. The polymer is moistened with the monomer, and the mass is allowed to stand for about five minutes. It is occasionally stirred, and when the mass is plastic, or about the consistency of soft putty, it is packed into one side of the mold. Wet cellophane is used as a separator between the two halves, and the flask is closed under heavy pressure. The two halves are again separated, and the excess is removed. Additional material is added, and the flask is again pressed together. Two testings are usually sufficient. The flask is then kept under pressure and is placed in cold water. The water is raised gradually to the

4. Sweeney, W. T.; Paffenbarger, G. C., and Beall, J. R.: Acrylic Resin for Dentures, J. Am. Dent. A. **29**:7 (Jan.) 1942.

5. This is known commercially as "film-ac" and is manufactured by Justi and Son, Inc., Philadelphia.

boiling point (about one-half hour) and then boiled one hour. The resulting plastic is usually clear and well cured. If bubbles are in the mass, it is the result of one of two conditions: lack of sufficient pressure and testing or raising the temperature to the boiling point too rapidly. In very thick masses it may be necessary to take as long as two hours to reach the curing temperature and then to cure the mass for five or six hours below the boiling point. This curing temperature may be as low as 160 F. with ten hours of curing. Collaboration with a dentist is advisable, since the technical construction of the implant is well understood by him.

In all of our cases, the defect had been traumatic in origin, and submucous resections had been performed previously. An outline of our operative procedure for the insertion of the implant follows.

OPERATIVE INSERTION OF THE IMPLANT

The patient's face and nose are surgically prepared with a tincture of a mercurial antiseptic containing a water-soluble dye as done in a submucous resection. The nasal mucous membrane is anesthetized with packs containing equal parts of epinephrine solution U.S.P. and 10 per cent cocaine hydrochloride in aqueous "zephiran chloride" 1:5,000. (The resulting solution is epinephrine hydrochloride 1:500 and cocaine hydrochloride 5 per cent.) Local anesthesia of the skin and subcutaneous tissues of the nose is obtained with a 2 per cent solution of procaine hydrochloride with 8 minims per ounce (0.5 cc. per 30 cc.) of epinephrine solution U.S.P. This infiltration is started at approximately the middle of the tip of the columella and extended over the entire dorsum of the nose with ballooning of the columella and the anterior extremity of the membranous septum. In approximately one half of our patients, however, anesthesia was induced with "pentothal sodium" without local infiltration.

The patient is lowered from a sitting to a reclining position, and, with Joseph's knife, an incision is made in the skin of the columella from the tip of the nose to the nasolabial angle. This incision is carried back to the cartilaginous septum, then extended dorsally above the upper lateral cartilages beneath the perichondrium, and on beneath the periosteum of the entire dorsum of the nasal bones. The previously fashioned methyl methacrylate splint is then inserted into the nose (fig. 1 *C* and *D*) with its upper extremity resting on the tip of the nasal bones and the tip of the base of the L-shaped portion resting on the nasal spine (fig. 1 *A* and *B*). The base or prop portion, in the anterior extremity of the septum, is anchored in place by a mattress suture of 000 chromic gut, which is passed through the septum and through the holes previously drilled in the base or prop. The cutaneous incision in the columella is closed with interrupted fine plastic sutures on an atraumatic needle. A splint of hard dental impression wax, molded in hot water, is then fashioned over the dorsum of the nose, care being taken to hold the implant in the desired position. With this in place, ice compresses are applied to set the molded stent, which is then anchored in place with adhesive straps. No nasal packs are necessary.

Immediately after the operation all patients did quite well except one who showed slight cellulitis of the nose. This cleared with hot compresses applied locally plus sulfadiazine by mouth, and the patient has retained the implant since October 1943, with good results. Eventual removal of the splint proved necessary in 6 of our 20 cases. A

casual glance at the statistics on our results might tend to lead one to discount the procedure, but we feel that when the causes of the failures are studied, the operation will be accepted as quite worth while and not condemned. Therefore, we briefly outline our failures:

The fourth patient received a methyl methacrylate implant on Oct. 4, 1943, which was to correct a depression deformity resulting from a crushing blow received at 6 years of age. The patient had had a submucous resection previously, plus six unsuccessful plastic procedures elsewhere. Six days after receiving the surgical implant he incurred a hard blow on the nose while playing with a child.

Fig. 1.—*A* and *B*, position of the implant in relation to the nasal structures; *C*, methyl methacrylate implant; *D*, insertion of the implant.

Then cellulitis of the nose and face developed, spreading rapidly, with a severe systemic reaction. The splint was removed.

Patient 7 engaged in a friendly boxing match seven months postoperatively and was struck on the nose. This caused the implant to protrude approximately one-quarter inch (6.5 mm.) through the skin of the tip of the nose. The exposed portion was removed with rongeurs, the tip was repaired, and the nose healed uneventfully, with a good cosmetic result. Eleven months later, however, following another blow, incurred in a fall aboard ship, a reaction developed over the bridge of the nose. This was incised and drained by the ship's physician. The lesion failed to heal, and the splint had to be removed, one and one-half years after its implantation. The nose then healed in its original depressed position. No further surgical correction is contemplated for this patient.

In patient 8 a stitch abscess developed in the anchor suture of the columella, which was inadvertently overlooked at the time of the removal of the sutures (the anchor suture in this patient was 000 silk). This progressed until there was a small sinus tract beneath the implant, which failed to heal in spite of the use of hot applications, sulfadiazine and penicillin, given locally and systemically. The splint had to be removed after five months. When seen three months later the cosmetic result was considered good. Apparently considerable scar tissue had developed, which supported the bridge of the nose.

In patient 10 a sinus tract developed, extending from within the left vestibule to beneath the implant. This was noticed in the first week and healed only after

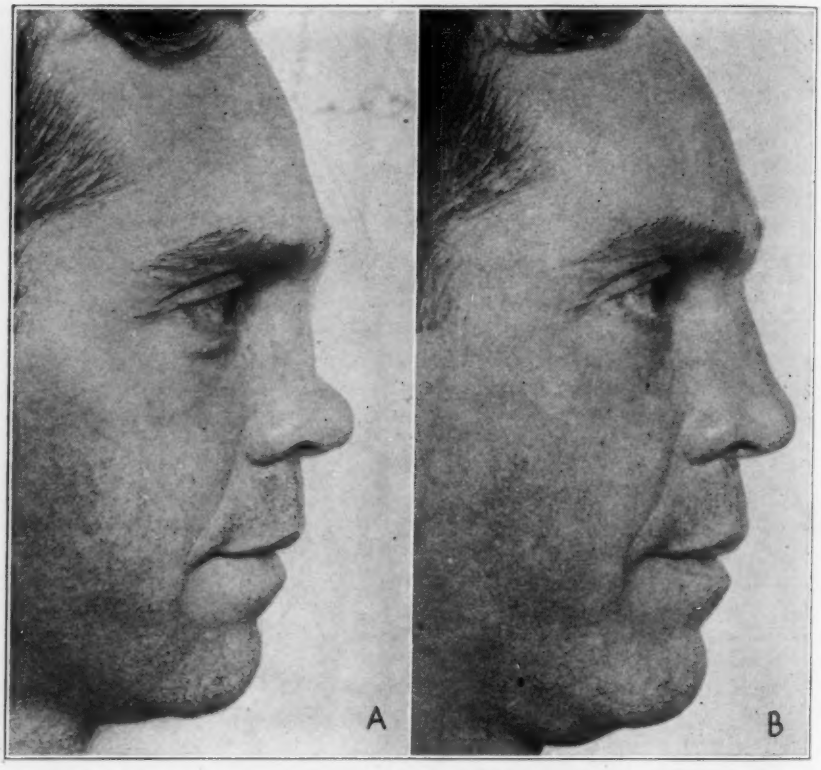

Fig. 2 (case 9).—*A*, depression deformity before surgical correction; *B*, appearance two months after rhinoplasty employing a methyl methacrylate implant.

the implant had been removed. The anesthesia was induced with "pentothal sodium." The patient had considerable scarring of the nose and columella from the original injury, which had been produced by a shell exploding in his face. This may have been a contributing factor in the failure.

Patient 11 was a 14 year old girl who required removal of the implant eight months after its insertion. She received a severe blow to the nose while playing basketball and soon afterward noticed a thin serous discharge in the left passage. On removal the implant was found to be broken into three pieces, one of which protruded into the vestibule. The nose then healed but returned to its original depressed position.

In patient 13, who had been under general anesthesia when operated on, a sinus developed, extending from the left vestibule to beneath the implant, with a slight serous drainage, soon after he was discharged from the hospital. This sinus was still present when he was seen six months later. The splint was removed after a surgical closure of the sinus was unsuccessful.

It would seem that these patients do not tolerate trauma well. However, the type of patient found in our series (seafaring men, especially merchant seamen) are more likely to become the victims of

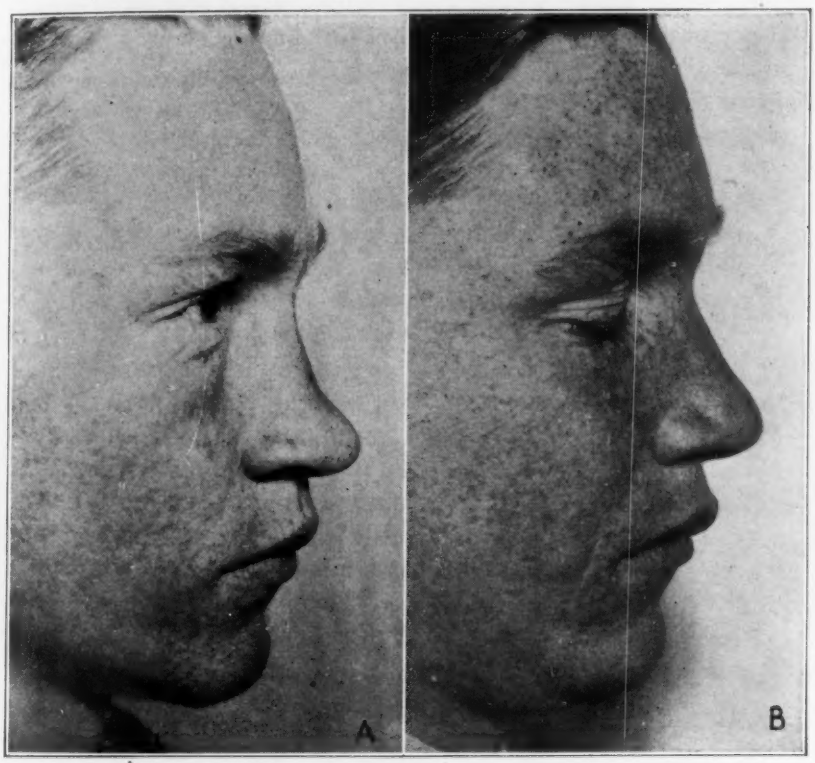

Fig. 3 (case 15).—*A*, depression deformity before surgical correction; *B*, appearance thirteen months after rhinoplasty with a methyl methacrylate implant.

injuries and less likely to exercise caution in protecting their noses and faces than the average patient seen in private practice. Patient 14 received such a severe blow to his nose that he was knocked to the ground, with resulting fracture of the nasal bones. His implant was loosened somewhat but retained its position, and the nose healed uneventfully with the splint retained. He still had a good cosmetic result when seen six months later.

We have not outlined our successes since all of the patients have what we consider to be good cosmetic results. Illustrations of typical

cases are shown (figs. 2 and 3). A point of interest which might be mentioned, but is not considered a handicap, is the fact that these patients complain of their noses becoming cold when exposed to low temperatures, especially in inclement weather. We believe that local anesthesia is preferable to general. This is because it facilitates making the original incision by ballooning the columella and by avoiding a communication extending into the vestibule. It also gives some hemostasis. We believe this procedure has definite advantages, but we are now testing a pliable plastic which, if satisfactory, would give more normal resiliency to the nose. We hope it lives up to expectations.

We wish to acknowledge the work of Dr. Leslie M. Smith, who is one of the originators of this method.

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TRAUMATIC LESIONS OF THE LARYNX

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IT HAS been roughly estimated that injuries of the larynx comprise about 1 per cent of military casualties, a figure which is probably too low, since most injuries of the neck are tabulated as such without any specific description of the type or the location. Many laryngeal injuries are diagnosed as multiple cervical injuries and consequently cannot be included in statistics of laryngeal injury. These mistakes are usually not corrected later—for, unfortunately, many of these wounds are fatal. Regardless of the low percentage of laryngeal injuries, they present one of the more serious forms of battle casualties not only from the aspect of immediate loss of life but also from the standpoint of repair of the larynx and rehabilitation of the patient.

Peacetime injuries of the larynx are rare and in the main differ greatly from wartime injuries. They usually result from industrial accidents, criminal attacks or suicidal attempts, either thermal or metallic, and are inflicted with sharp instruments or a direct or an indirect blow. The injury is usually much less extensive and severe, and there is little or no loss of tissue. Combat injuries are usually made by a direct sharp metallic instrument, which may be smooth, like a rifle bullet, or rough and jagged, like shrapnel. For this reason these injuries are more extensive, and there is often considerable loss of tissue.

The first concern is the immediate first aid treatment, which is given at the place of injury or at the nearest aid station. The proper care of the injured at this time is of utmost importance, since great damage can be done by improper handling of the patient. The two main essentials are to secure an adequate and dry airway and to control hemorrhage. The control of hemorrhage is usually handled competently and efficiently, but the management of the airway is another matter. Far too frequently high tracheotomy is performed in the aid station and in the civilian hospital. It is true that tracheotomy is a life-saving procedure, and the immediate concern is to provide an adequate airway, but low tracheotomy

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From the Department of Otolaryngology, Tulane University School of Medicine, and the Department of Otorhinolaryngology, Ochsner Clinic.

can be accomplished with little more detail of procedure and much less possibility of later development of stricture and deformity than can high tracheotomy. In many instances low tracheotomy has been done on the battlefield with good results and has required no follow-up care in the general hospital; so there is no doubt that low tracheotomy can be done as a military or a civilian emergency. If necessary, a tracheotomy tube may be inserted into the laryngeal or the tracheal wound after the bleeding has been controlled, and proper tracheotomy can be done at the field, the station or the civilian hospital later.

Since the patients are usually in a state of shock, proper precautions should be taken against further shock, and measures to combat the immediate shock should be instituted. Gunshot and especially shrapnel wounds of the larynx produce a state of shock out of all proportion to the injury and differ somewhat in this regard from peacetime laryngeal trauma. Shock may be overcome by the administration of a plasma infusion, by the application of heat and by reassurance of the patient. Sedatives, especially opiates, are contraindicated because they tend to decrease the respirations and depress the cough reflex.

After an adequate airway has been provided, hemorrhage has been controlled and shock combated, the extent of injury should be determined. Injuries may be divided into those situated above the hyoid bone, those involving the thyroid cartilage, those involving the cricoid cartilage and those of the trachea. This classification is applicable to stab wounds, gunshot wounds and blows on the neck but not to shrapnel wounds, which usually involve several structures and sometimes the entire anterior portion of the neck. Wounds of the frontal surface of the neck, if no damage has been done to the cartilage, should be débrided and sutured. However, with sulfonamide drugs being implanted in the wound and extensive use made of penicillin, débridement can be reduced to a minimum, and as much tissue as possible should be preserved. The structures encountered in this location are so compact and so closely related that extensive removal of tissue often does more harm than good. A simple gunshot wound that has been subjected to extensive débridement may result in paralysis of the cricothyroid muscle through injury to the external branch of the superior laryngeal nerve and, as a result, a simple wound of the neck is converted into a complicated one. Wounds involving the frontal surface of the neck heal without undue complications, and the prognosis is good.

Next in severity are open or subcutaneous wounds of the thyroid cartilage. The prognosis as to voice and breathing is good, and recovery usually ensues. However, the patients should be examined repeatedly for possible laryngeal edema, which they all have at first, and for hematoma, which may interfere with the airway. They should also be

watched carefully for the onset or the presence of chondritis or perichondritis, which is not easy to detect, since the only positive finding is slight tenderness, which may be persistent and sometimes progressive. The sedimentation rate, the blood picture and the temperature remain normal. There has been much controversy as to the time and the advisability of operation in these cases. It has been my experience that more of the cartilaginous structures can be preserved and maintained by operating early, with drainage and chemotherapy, than by delaying operation.

Shattering of part of the cricoid cartilage presents a serious problem. There is apt to be not only web formation but complete cicatricial stenosis of the larynx, and with loss of support there is collapse of the larynx at this point, which may be extensive and severe. The stenosis is much more extensive and serious than that seen in cases of high tracheotomy, and the prognosis is grave.

If the shrapnel is of large size or of low velocity, there may be impairment of the integrity of all the laryngeal cartilages. This comprises the next thing to complete excision of the larynx and frequently is fatal. The establishment of an adequate airway, necessitating many operative procedures, plus the teaching of phonation requires all the ingenuity of the surgeon and taxes the disposition of the injured.

Another more extensive injury is that involving one or two tracheal rings, the cricoid and the thyroid cartilage and the hyoid bone. The only difference between this injury and the one described in the preceding paragraph is in the extent of the injury; the operative procedure is the same if it is possible to attempt plastic repair. This injury, however, is usually fatal at the site at which it is incurred, and rarely if ever does the subject reach the hospital for treatment. This same injury may be complicated by injury of the cervical vertebrae, which also is usually fatal. In the latter three types of injury the esophagus is usually also damaged to a greater or less extent, and esophageal damage has been recorded in practically every case. This was true in those cases in which the patient reached our general hospital during World War II.

The diagnosis of laryngeal injury can be made on external inspection, as the wound is usually only partially healed by the time the patient reaches the hospital, or there is persistent tracheotomy because of narrowing of the lumen. In the presence of an open wound, careful inspection is necessary to determine not only the extent of the injury and loss of structure but, more important, the amount of tissue and cartilage remaining as a basis on which to build. No structure which appears to have the least chance of survival should be removed, and it is essential to preserve as much tissue and cartilage as possible. It is always important to determine whether the esophagus has been injured and whether

or not there is a fistula. If the esophagus has been injured, it is important to establish first whether the lumen, and second whether the airway, is adequate. If the wound is closed, a roentgenogram made prior to direct examination will help determine how much cartilage remains and whether there has been a fracture of the cartilages or of the hyoid bone. The symptoms of fracture of the hyoid bone are expectoration of blood, injury of the pharyngeal mucosa, disturbance of phonation, pain when the tongue is extended or moved from side to side, and immobility of the epiglottis. However, since by the time the patient reaches a general hospital the symptoms may have subsided, a roentgenogram should be made to determine the possibility of injury. Indirect, or mirror, examination will enable the examiner to see many points which may be noted and later recalled when the direct examination is done. Following the external and the indirect examination every patient should have a direct examination; for this I prefer suspension laryngoscopy, for in no other way may one sit down and take time, with both hands free, to make a careful examination. The mouth, the base of the tongue, the hypopharynx and the opening of the esophagus are brought into view. Then, the arytenoid structures, the ventricular bands, the ventricles and the vocal cords are examined. The cords may be retracted and the area beyond studied more conveniently and as thoroughly as with any other method. Fixation of the arytenoid cartilages, paralysis of one or both cords, webs, cicatricial bands and the degree of stenosis can be noted.

After the complete external examination and the indirect and finally the direct laryngoscopic examination have been made, repair of the injuries may be undertaken. Esophageal injury should receive first attention. If the injury is small and a fistula is present, this should be excised and the wound closed with a feeding tube left in for from five to seven days. If the injury is extensive, then gastrostomy should be done prior to repair of the defect. After closure of the esophagus, an adequate lumen should be assured before laryngeal repair is attempted. If the lumen is not adequate, dilation should be done and an adequate passage firmly established before proceeding further.

In regard to the larynx, low tracheotomy should be the first procedure if it has not already been done. An attempt should be made to go as low as possible, so as to get as far away from the injury as feasible, and to have the patient fully accustomed to the tracheotomy tube before proceeding further. If a web is present, a stint should be inserted as far anteriorly as possible, and the tissue around this area should be allowed to heal before the web is excised; otherwise it will have a tendency to recur. If there is a stricture with a lumen and the scar is not too thick or too extensive, it may yield to repeated dilations. If, however, the scar tissue happens to be dense and thick, then a stint

or a mold should be inserted, the size of mold being gradually increased as the scar tissue yields and retracts. For this tantalum molds of various sizes should be used, as molds made of this substance have been found less irritating, are well tolerated by the larynx and are relatively comfortable to the patient after being once inserted. Dense scar tissue producing complete stricture, if thin, can be incised during suspension laryngoscopy, and then tantalum molds can be employed. However, if the scar tissue is dense and the stricture of some extent, laryngofissure should be done, the scar excised and a skin graft inserted. Following this, molds and dilations are necessary to provide a complete and adequate airway. Loss of part of the thyroid cartilage and especially of the cricoid cartilage requires that cartilage be transplanted from the rib in order to maintain an adequate lumen; unless this is done, there is a tendency toward collapse, especially on movement of the head, and the lumen will suddenly close down or the airway will be completely blocked off.

Complications are more prone to result from laryngeal injuries than from injuries of most other parts of the body. The fascia spaces provide pathways for infections of the neck, extending to the mediastinum; pneumonia may develop from aspiration, and emphysema, from a too closely sutured wound. Because of the mucous glands of the trachea, the skin graft, as a foreign body, becomes wet and is liable to slough, and secondary infection of the operative site within the larynx may follow. Chondritis and perichondritis are ever present hazards, and, if they occur, their sites should be drained at once to preserve as much cartilage as possible. These complications may be guarded against and combated by giving transfusions, by administering sulfonamide compounds and penicillin both before and after operation and by maintaining a closed respiratory system during anesthetization and operation so that no blood or secretions may enter the trachea or the bronchi.

Of final importance in treatment is the rehabilitation of the patient. The function of the larynx is twofold—that of respiration and that of phonation. Much work has been done in regard to the former, but only lately have schools been established and teachers trained for helping persons to improve phonation. If no attempt is made to improve or correct phonation, a severe psychosis may develop. Breathing is not enough; the patient also wants to talk. Subsequent to the completion of surgical treatment and the establishment of an adequate airway, the patient should be sent to a speech center before his final discharge.

Of 38 patients with laryngeal injuries treated during World War II, from November 1943 to June 1946, all but 2 have been decanalized and returned to duty or discharged to civilian life. These 2 patients will require additional treatment before the final stage can be reached, if ever.

SUMMARY

It is difficult to estimate the incidence of laryngeal injuries, because many are classified under cervical injuries. There are three main phases of the care of this type of injury. First is the immediate care, given on the battlefield or in the dressing station, which includes low tracheotomy and supportive treatment to combat shock. If treatment must be postponed for an indefinite period, low tracheotomy should be done immediately in all cases of laryngeal injury. The second phase consists of plastic repair, dilations and attempts to establish an adequate airway. The last phase is the rehabilitation of the patient. After the completion of surgical treatment and the establishment of an adequate airway, the patient should be sent to a speech center before final discharge.

ABSTRACT OF DISCUSSION

JOSEPH A. BUCHIGNANI, Memphis, Tenn.: I wish to elaborate on the social problem that arises from this unfortunate circumstance. During the process of plastic repair of severe traumatic lesions of the larynx, there exists, as Dr. Lynch has pointed out, the possibility and likely the probability of a failure to produce the desired surgical result, and sometimes what might be a factor more discouraging to the continued efforts of the surgeon, that frequently encountered obstacle, the patient's lack of will to live. This same obstacle can occur during the process of rehabilitation.

This does not resolve itself into a problem of segregation, but rather of association with similarly afflicted persons, which gives impetus to the spirit of competition, the basis of attainment of any goal.

I suggest that any person so afflicted would receive much more benefit from his surgical and rehabilitative care if he were living, eating, thinking and learning among others of his kind. The synergistic effect of this type of convalescence could prove to be invaluable.

DR. J. WADE YOUNGBLOOD, Dallas, Texas: Since the cessation of warfare, it is not likely that otolaryngologists are going to be called on, except in exceptional cases, to treat traumatic lesions. There are traumatic lesions of the larynx produced by poor surgical technic, the use of caustic acids and alkalis and overirradiation that are just as serious to the patient and cause as much concern to the laryngologist as the lesions described by Dr. Lynch.

The most common cause of perichondritis with resultant stenosis of the larynx is high tracheotomy. Tracheotomy is considered too often only as a life-saving procedure, one that must be done immediately, with the patient near death. A hasty tracheotomy is usually made high, causing perichondritis, with resulting cicatricial formation, and owing to the fact that there is no resistance to this scar formation, the lumen of the larynx is completely obliterated. If tracheotomy has been done high in haste or through error, the operation should be repeated in a lower position as soon as the patient is able, with decanalization of the upper opening.

The larynx, as any injured part of the body, will respond most satisfactorily to rest. The patient has everything to lose and nothing to gain by waiting for the procedure to become an emergency. If there are dyspnea, pallor and retraction of the suprasternal notch, certainly tracheotomy should be performed. It has been my experience that the rest afforded the larynx by tracheotomy results in much quicker healing and more comfort to the patient, as well as saving the physician

anxiety. Even though high tracheotomy is the most common cause of surgical trauma of the larynx, tracheotomy is a useful operation when performed as early as indicated, and low.

Other traumatic lesions of the larynx resulting from treatment are burns from galvanocautery, caustic acids and alkalis or from irradiation. The latter is mentioned only to stress the effects of irradiation on thyroid cartilage. The laryngeal cartilages are particularly sensative to irradiation, and ultimate destruction of cartilage may result from overdosage. In burns of the mucous membranes of the larynx, there is edema, which may subside with little trouble if mild, or there may be necrosis with sloughing and ulceration and subsequent scar formation according to the severity of exposure. In irradiation of the larynx, there is edema, but very seldom necrosis and sloughing immediately following the exposure.

This initial edema may subside, or it may remain as a chronic edema for weeks or months, with recurrence, which may be accompanied with sloughing and necrosis due to breaking down of the thyroid cartilage. Overdosage of radiation causes perichondritis and radionecrosis particularly in the inferior portion of the thyroid cartilage. Since there are some large blood vessels in this area, it is not unusual for them to be involved in this necrosis, and this may cause severe hemorrhage and even death unless readily controlled. This is not to say that irradiation, galvanocautery or caustic acids and alkalis should not be used properly; traumatic lesions of the larynx may occur as a result of their use. One is certainly justified in using anything that offers hope of arresting the life-menacing cells in inoperable carcinoma of the larynx.

Dr. G. E. Martin, Edinburgh, Scotland: I am glad to be allowed to enter this discussion. During and after the recent war many traumatic injuries of the larynx have come under my care. I do not think Dr. Lynch has emphasized sufficiently the imminent danger of many of these injuries, especially gunshot wounds. I have seen within the last year 3 cases of carcinoma—1 in which the larynx and 2 in which the hypopharynx was involved—following perforating gunshot wounds of the larynx. In 2 of these the wounds were received in World War I. In that war many simple perforating wounds of the larynx or the hypopharynx, once the immediate danger was over, were left to heal by themselves. I am afraid it was forgotten that epithelial cells could be carried in with the bullet or the shrapnel, and though there was only a small scar left on the surface it gave rise later to an epithelial tumor of the underlying mucous membrane.

In the blackout there were, unfortunately, a number of bag-snatchings. The assailant very often gripped his victim by the throat. In 2 recent cases this actually caused a dislocation of the arytenoid cartilages. Such a dislocation does not seem to do any harm; it gives rise to a certain amount of huskiness but with suitable vocal exercises, started as soon as possible, the condition leaves no ill effect.

Dr. Henry B. Orton, Newark, N. J.: I agree with what Dr. Martin has stated. In the City Hospital in Newark my associates and I are closely associated with the Examiner's Office, and we get a great many of these external injuries of the neck and larynx. Our treatment has been much as Dr. Lynch stated, airway first, and stop the hemorrhage later, with careful suturing of individual structures separately.

Just before I came away, we had to deal with a gunshot wound of the neck. The bullet had penetrated the carotid artery and had come out through the back of the neck. The wound closed over, and the patient was admitted to the hospital as paralytic. The bullet had gone through the carotid artery, with a plug being left in the internal carotid artery, and then had caused the paralysis.

I have observed 2 cases of carcinoma of the larynx due to effects of mustard gas inhaled in the first war. Dr. Martin brought out the question of plugging, which has been quite prevalent as a cause of edema of the retina. The rehabilitation of patients who have undergone this injury must take place very quickly; otherwise, their disability becomes chronic, and the same observation applies to all persons who have to be rehabilitated, even those who have undergone laryngectomy, that they do far better if they are in a class of patients with injuries of the same sort.

DR. E. G. GILL, Roanoke, Va.: Dr. Lynch mentioned that one of the most frequent causes of hemorrhage in emergency tracheotomies is a surgical operation improperly performed. I agree. This complication can be prevented by the use of the laryngoscope and the bronchoscope. After the bronchoscope has been introduced, the emergency is over. One has ample time to do a deliberate and careful tracheotomy.

NONREGENERATION OF THE MUCOUS MEMBRANE OF THE FRONTAL SINUS AFTER ITS SURGICAL REMOVAL (IN THE DOG)

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WHAT occurs in the paranasal sinuses after surgical removal of the mucous membrane has been a controversial subject for many years. McGregor,[1] of Toronto, found a good deal of regeneration in 26 specimens taken from the antrum, stating that perfectly reformed mucous membrane was present. He went on to say that it grows by inundation and transplantation and that bone itself may produce connective tissue.

Coates and Ersner[2] found regenerated mucous membrane in the frontal sinus of a dog two months and seven months after surgical removal of the membrane.

Gorham and Bacher,[3] of San Francisco, observed remarkable regeneration in the human maxillary antrum in from three to five months after operation in most cases, and certainly in ten months in others. They noted that ciliated columnar epithelium with loose stroma, serous and mucous glands and a definite basement membrane were present.

Unfortunately, in our experimental work on the frontal sinus of the dog we were not able to duplicate any of these utopian results; in fact, we found the exact opposite to be the prevailing picture.

Moreover, several others found a distinct lack of regeneration in the paranasal sinuses. Kistner,[4] of Portland, Ore., in a study based

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This article was read before the Philadelphia Laryngological Society on Nov. 4, 1947.

From the Department of Otolaryngology and the Harrison Department of Surgical Research, University of Pennsylvania School of Medicine.

1. McGregor, G.: Further Proof of Regeneration of Mucous Membrane in Antrum, Arch. Otolaryng. **14**:309 (Sept.) 1931.

2. Coates, G. M., and Ersner, M.: Regeneration of the Mucous Membrane of the Frontal Sinus After Its Surgical Removal (in the Dog), Arch. Otolaryng. **12**:642 (Nov.) 1930.

3. Gorham, C. B., and Bacher, J. A.: Regeneration of Human Maxillary Antral Lining, Arch. Otolaryng. **11**:763 (June) 1930.

4. Kistner, F.: Histopathology and Bacteriology of Sinusitis: Postoperative Repair, Arch. Otolaryng. **13**:225 (Feb.) 1931.

on 15 persons, in whom the Caldwell-Luc and similar operations were done, found only a thick deposit of organized scar tissue in the antrum. He claimed that the new lining in an antrum is not regenerated epithelium but takes its origin from the nasal or the oral epithelium, whichever is unintentionally pushed in or grows in.

Rosenow, of New York, after extensive study of postoperative sinal membrane, came to the conclusion that it does not regenerate but appears in the form of scar tissue and epithelium resembling that in the area from which it comes.

With all the foregoing statements and data in mind, we decided to use the frontal sinus of the dog and, by operating on a fairly large number of dogs, try to elucidate this question of regeneration of mucous membrane.

Under aseptic precautions, we operated on approximately 10 dogs over a period of one year, removing the mucous membrane from the frontal sinus, and opening the sinus two weeks, one month, two months, three months and four months later to observe the results. Four of the dogs died of intercurrent infections; the remaining 6 stood the operative procedures well and were in good condition at the end of the year.

We did numerous operations on the frontal sinuses of the dogs. Sections of the normal mucosa were removed at the first operation, as a control.

Figure 1 shows a section of normal mucous membrane under low and high power magnification. This particular section was taken from the right frontal sinus of dog 310, operated on Nov. 22, 1946. On December 30 (a little over one month later) the right frontal sinus was reopened and found markedly diminished in size—about one-third the size of the original sinus. The remainder of the sinus was occupied by generally encroaching bone and thickened lining, the latter approximately 3 mm. thick; no infection was present, and the dog was in good condition. A specimen of the lining was taken and is shown in figure 2. It is obvious that there was no fine columnar epithelium with formed folds or papilla-like projections. In fact, there was no regenerated mucosa at all; the lining of the sinus was composed entirely of fibrous tissue without evidence of inflammation. Within the depths of the tissue the fibrous stroma was relatively dense and avascular; however, fibroblasts and capillaries were still proliferating.

On March 7, 1947, dog 696 was operated on, normal mucosa removed from the right frontal sinus and the wound closed. On May 9 the right frontal sinus was found completely filled with soft fibrous tissue. This was removed in its entirety, and specimens sent for section. The bony walls were found rough and nodular, and the size of the cavity was about three-quarters that of the left frontal sinus, which was opened and found normal in all respects.

Fig. 1.—Normal mucosa of the right frontal sinus of dog 310: *A*, low power magnification; *B*, high power.

The section is shown in figure 3. Again there was no evidence of epithelium or mucosa; the lining of the sinus was composed principally of dense fibrous tissue without inflammatory exudate. There were minute areas of incompletely calcified osteoid tissue embedded in the fibrous stroma.

In some areas of the fibrous tissue were scattered numerous atypical striated muscle fibers, these fibers having no definite pattern or organization. The nuclei were often centrally located and were frequently

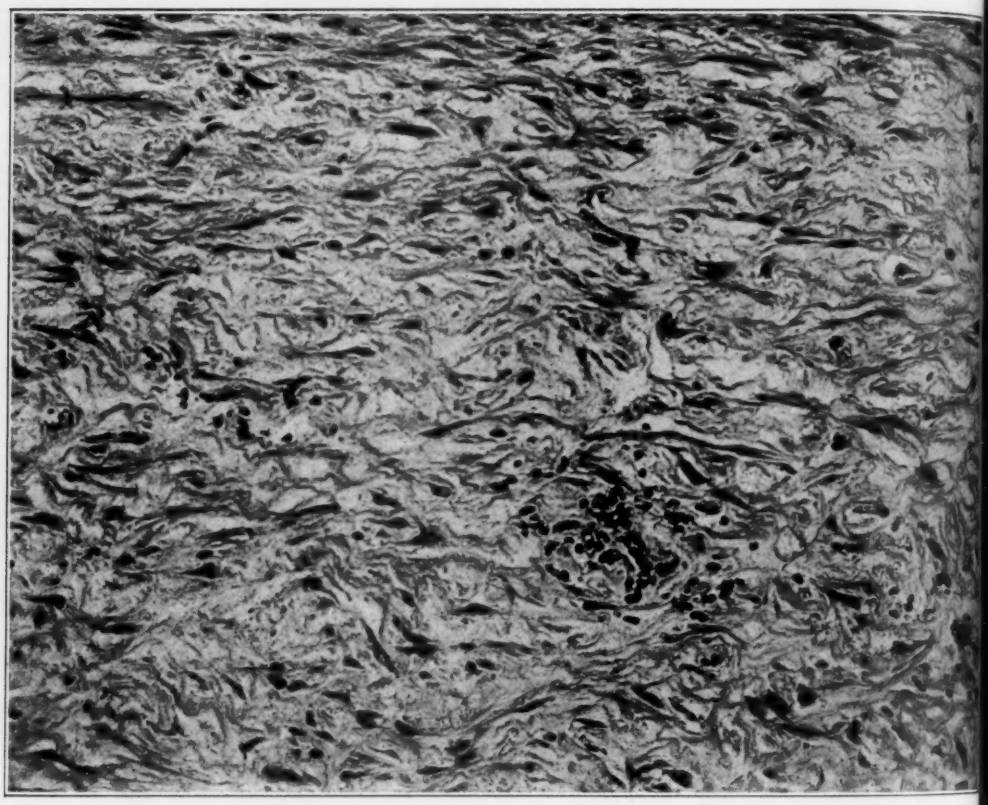

Fig. 2.—Fibrous tissue lining the right frontal sinus of dog 310 one month after surgical removal of the mucous membrane.

multiple; with the Van Gieson stain both the fibrils and the cross striae were visible in many of the fibers. This resembled regenerating muscle and did not appear neoplastic.

On Feb. 25, 1947, we operated on dog 628 and removed the normal mucosa of the right frontal sinus. The animal lost weight and energy and appeared emaciated; however, there was no evidence of localized infection around the operative site. The sinus was reopened and examined again on May 23 (three months later) and was found to have been completely obliterated by heavy fibrous connective tissue.

Sections were taken, and examination disclosed very dense fibrous tissue, similar to that seen in figure 3.

On Jan. 10, 1947, dog 144 was operated on, the right frontal sinus mucosa curetted out, specimens taken and the wound closed after complete hemostasis. This dog remained in good health and was again operated on four months later, on May 9; on incising the site of the right frontal sinus, we found no cavity whatsoever. Removal of the fibrous tissue left a bony cavity 1 cm. in diameter in communication

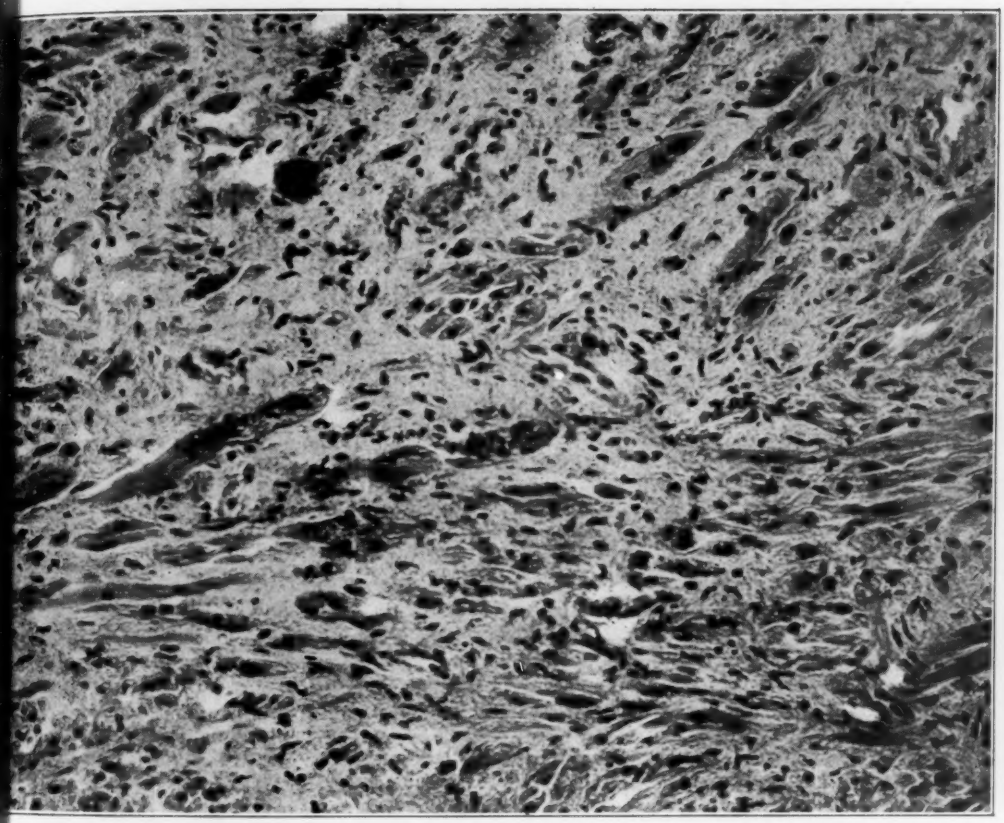

Fig. 3.—Fibrous tissue, muscle strands and other tissue elements lining the right frontal sinus of dog 696 two months after surgical removal of the mucous membrane.

with the nasofrontal duct, which seemed to be patent. A sample of the fibrous tissue was sent for section. The left frontal sinus, opened for comparison, was found to be of normal size and lined with delicate mucous membrane.

Figure 4 shows the tissue observed in the right sinus of this dog at four months: The bulk of the tissue was likewise fibrous in character without inflammatory exudate. The areas of incompletely calcified

osteoid tissue were greater in extent, and there were small foci of hyaline cartilage embedded in the fibrous stroma. There was no microscopic evidence that this cartilage was being converted into osteoid tissue. In fact, the cartilage appeared to be degenerating, since most of the cells had lost their nuclei.

COMMENT

The lack of regeneration of mucous membrane in the surgically treated frontal sinuses of dogs was striking. No inflammatory exudate

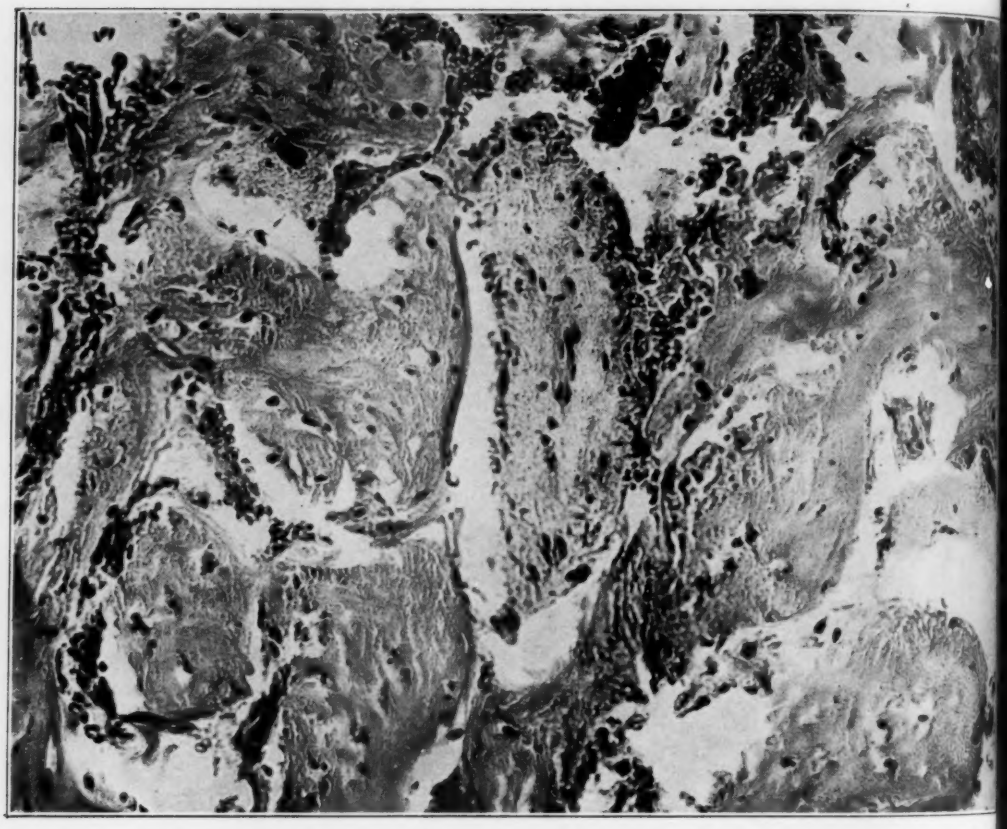

Fig. 4.—Osteoid tissue seen in the right frontal sinus of dog 144 four months after removal of the mucous membrane.

was seen in any specimen. What was present was a fibrous tissue process, with new bone and cartilage probably coming from the periosteum.

The atypical muscle strands may be due to inundation from the supraorbital muscle at operation and probably do not occur a priori.

Also, we might speculate that islands of mucous membrane were left behind inadvertently by the investigators who found regeneration.

We were careful to remove all of the mucous membrane and never found any regeneration whatsoever.

This work may explain some of the poor results which have so often followed radical or extensive operations on the paranasal sinuses in the human being.

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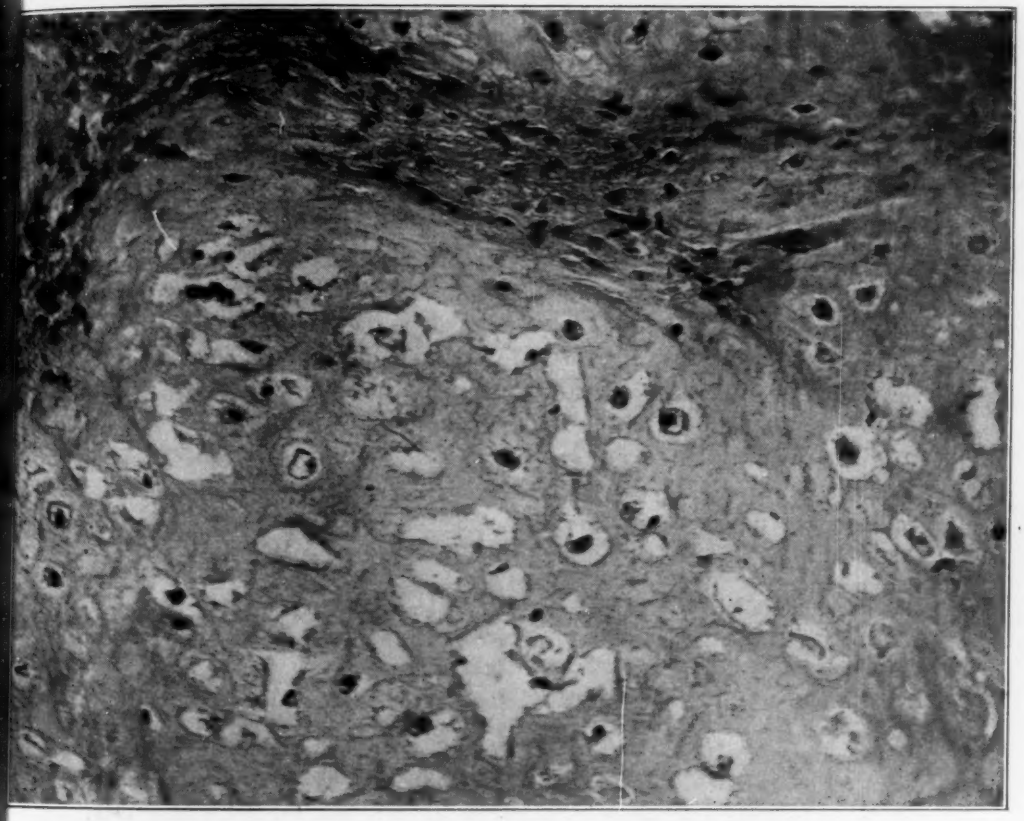

Fig. 5.—Cartilage seen in the right frontal sinus of dog 144 four months after surgical removal of the membrane.

ACKNOWLEDGMENTS

Dr. I. S. Ravdin gave assistance and advice in the carrying out of this work and the preparing of specimens. Dr. H. P. Schenck, professor of otolaryngology, University of Pennsylvania, gave encouragement and excellent advice with respect to the entire problem. Dr. R. F. Norris, of the Pepper Laboratory, University of Pennsylvania, contributed a detailed study and report on the microscopic sections. Mr. B. B. Varian prepared the photomicrographs of the microscopic sections.

CHORDA TYMPANI NERVE GRAFT

A Preliminary Report of a New Technic Used in Surgical Fenestration of the Labyrinth

SAMUEL ROSEN, M.D.
NEW YORK

OTOLOGISTS owe to Lempert [1] the first practical one stage technic for the surgical treatment of clinical otosclerosis. However, significant numbers of failures continue to be a challenge to the surgical technics developed therefrom. The two most important causes of failure appear to be (*a*) bony or fibrotic closure of the fenestra and (*b*) postoperative membranous labyrinthitis of varying degrees. Analysis of these factors has led Lempert [2] and Shambaugh [3] to introduce modifications of the fenestration technic in an attempt to increase the percentage of successful cases. Possibly the amount of auditory improvement could be increased to an even higher level in successful cases if the degree and the duration of the postoperative labyrinthitis could be diminished.

The fenestration operation makes greater demands of technical efficiency on the surgeon than most surgical procedures. The manipulations are unusual, and the consequences of small errors are more devastating. These technical difficulties can best be overcome by

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From the Otolaryngological Service, Mount Sinai Hospital.

Read at the meeting of the Section on Otolaryngology of the New York Academy of Medicine, Jan. 21, 1948.

1. Lempert, J.: Improvement of Hearing in Cases of Otosclerosis: New, One Stage Surgical Technic, Arch. Otolaryng. **28**:42-97 (July) 1938.

2. Lempert, J.: Fenestra Nov-Ovalis: New Oral Window for Improvement of Hearing in Cases of Otosclerosis, Arch. Otolaryng. **34**:880-912 (Nov.) 1941; Lempert Fenestra Nov-Ovalis with Mobile Stopple, ibid. **41**:1-41 (Jan.) 1945; Lempert Fenestra Nov-Ovalis Operation for the Restoration of Serviceable Unaided Hearing in Patients with Clinical Otosclerosis: Its Present Evolutionary Status, ibid. **46**:478-511 (Oct.) 1947.

3. Shambaugh, G. E., Jr.: The Surgical Treatment of Deafness, Illinois M. J. **81**:104-108 (Feb.) 1942; Surgical Treatment of Otosclerosis: Comparative Analysis of Results Using Different Techniques, Ann. Otol., Rhin. & Laryng. **51**:817-829 (Sept.) 1942; The Fenestration Operation for Otosclerosis, J.A.M.A. **130**:999-1006 (April 13) 1946. Shambaugh, G. E., Jr., and Juers, A. L.: Surgical Treatment of Otosclerosis: Preliminary Report on New Fenestration Technic, Arch. Otolaryng. **43**:549-567 (June) 1946.

practicing extensively on cadavers.[4] A large experience of this type has afforded me opportunities for observing a wide range of anatomic variations and for experimenting with variations of technic.

In this paper I shall describe a new procedure which I have introduced in a small series of cases, together with the preliminary results, which have been sufficiently encouraging to warrant a wider trial of the procedure.

TECHNIC

The chorda tympani nerve is liberated from its attachment to the facial nerve and is placed over the fenestra to protect the membranous labyrinth and the perilymphatic space from the untoward effects of blood and inflammatory products coming mostly from the overlying tympanomeatal flap. The chorda seems eminently suited to play the role of protective barrier to the delicate labyrinth since it withstands successfully all degrees of acute purulent otitis media to which it is exposed. Taste on the anterior two thirds of the tongue is not affected in acute purulent otitis media. It is interesting to note that in its tympanic course the chorda tympani nerve is covered with mucous membrane.

In every radical mastoidectomy the chorda tympani nerve is destroyed. During the course of the fenestration operation the chorda tympani is occasionally accidentally torn. In neither instance does the patient notice the loss of taste on the anterior two thirds of the tongue. In discussing all aspects of the operation with patients, I tell each one of the possibility of some loss of taste. No patient, however, has reported such loss.

A myringotomy knife is inserted between the chorda tympani nerve and the inner surface of the tympanic membrane. The blunt edge of the knife is swept upward beyond the point at which the chorda is attached to the neck of the malleus. In a similar manner the myringotomy knife is swept downward to the region where the chorda enters its bony canal (fig. 1*A*). The chorda, covered by mucosa, is then grasped with a jeweler's tweezers about 3 to 5 mm. below the malleus. Traction upward with the tweezers is continued (fig. 1*B*) until the chorda is pulled out of its bony canal (fig. 1*C*). The free length of the chorda is then placed safely in the tympanum until the fenestra nov-ovalis is completed. Then the chorda is placed over the middle of the length of the fenestra from above downward. In this position the chorda tympani lies on the delicate membranous labyrinth with only the very slightest perilymphatic space visible on each side of the nerve (fig. 1*D*). As the chorda is being pulled out of its canal great care is necessary to avoid tearing it before its free end is long enough to reach and cover the fenestra. When the chorda has been completely evulsed from its attachment to the facial nerve (fig. 1*E*) it may be long enough to be looped back up alongside and parallel to the descending portion of the chorda. This double length of chorda tympani covers the fenestra completely (fig. 1*F*). The tympanomeatal flap is then put in place.

4. Rosen, S.: Learning the Lempert Fenestration Operation, Arch. Otolaryng. **45**:335-338 (March) 1947. Maxwell, J. H.: Training of Surgeon and Selection of Patient for the Fenestration Operation, ibid. **46**:539-543 (Oct.) 1947.

REPORT OF CASES AND CLINICAL DATA

The chorda tympani graft described was first performed on Feb. 5, 1947. The surprisingly favorable early return of useful hearing has resulted in my applying this technic in all later cases. There has been no arbitrary selection of cases. The new procedure has been attempted in every instance in which fenestration has been performed since February. It has been successful in each of the 8 cases reported in this paper. In 2 cases the chorda tympani was torn and could not reach the fenestra. These 2 cases are not included in this report.

CASE 1.—F. W., a 23 year old single woman, had mild diabetes. Progressive deafness had been noted in both ears for three to four years. There was no family history of deafness. The operation was performed on Feb. 5, 1947. Audiograms taken at various stages are shown in figure 2.

CASE 2.—N. R., a 37 year old married woman with two children, had noted progressive deafness for seventeen years. It increased following pregnancies. There was no family history of deafness. The operation was uneventful. On the sixth postoperative day a large black blood clot was seen under the entire tympanomeatal flap. The clot filled the tympanum and extended posteriorly over the fenestra and for 6 to 7 mm. behind it. For two weeks hearing in this ear was reduced to perception of moderate voice close to the ear. At the end of this time the blood clot disappeared from the tympanum but persisted under the rest of the flap over the fenestra for more than two weeks longer. Then it gradually disappeared. Audiograms are shown in figure 3.

CASE 3.—P. S., a 40 year old woman, had noted progressive deafness for the past ten years. She completed her first full term pregnancy five months before operation. The fenestration and the postoperative course were uneventful.

CASE 4.—R. T., a 37 year old woman with two children, had noted progressive deafness for ten years. It had been accelerated since her last pregnancy. The fenestration and the postoperative course were uneventful.

CASE 5.—M. B., a 36 year old man, had noted progressive deafness for the past five years. One brother was similarly affected. Separating the meatal skin close to the drum resulted in a small rent so that saline solution poured into the membranous canal above ran out below. The 2 mm. rent in the meatal flap was about 2 mm. from the fenestra. The postoperative course was uneventful. Audiograms are shown in figure 4.

CASE 6.—M. L., a single woman of 23 years, had noted progressive deafness for the past four years. There was a history of familial deafness. The fenestration and the postoperative course were uneventful.

CASE 7.—B. A., a widow of 30, with 1 child, had noted progressive deafness for the past six to seven years. There was a history of familial deafness. The fenestration and the postoperative course were uneventful.

CASE 8.—D. S., a 46 year old woman, had experienced progressive deafness for twelve years. There was a history of familial deafness. The fenestration and the postoperative course were uneventful.

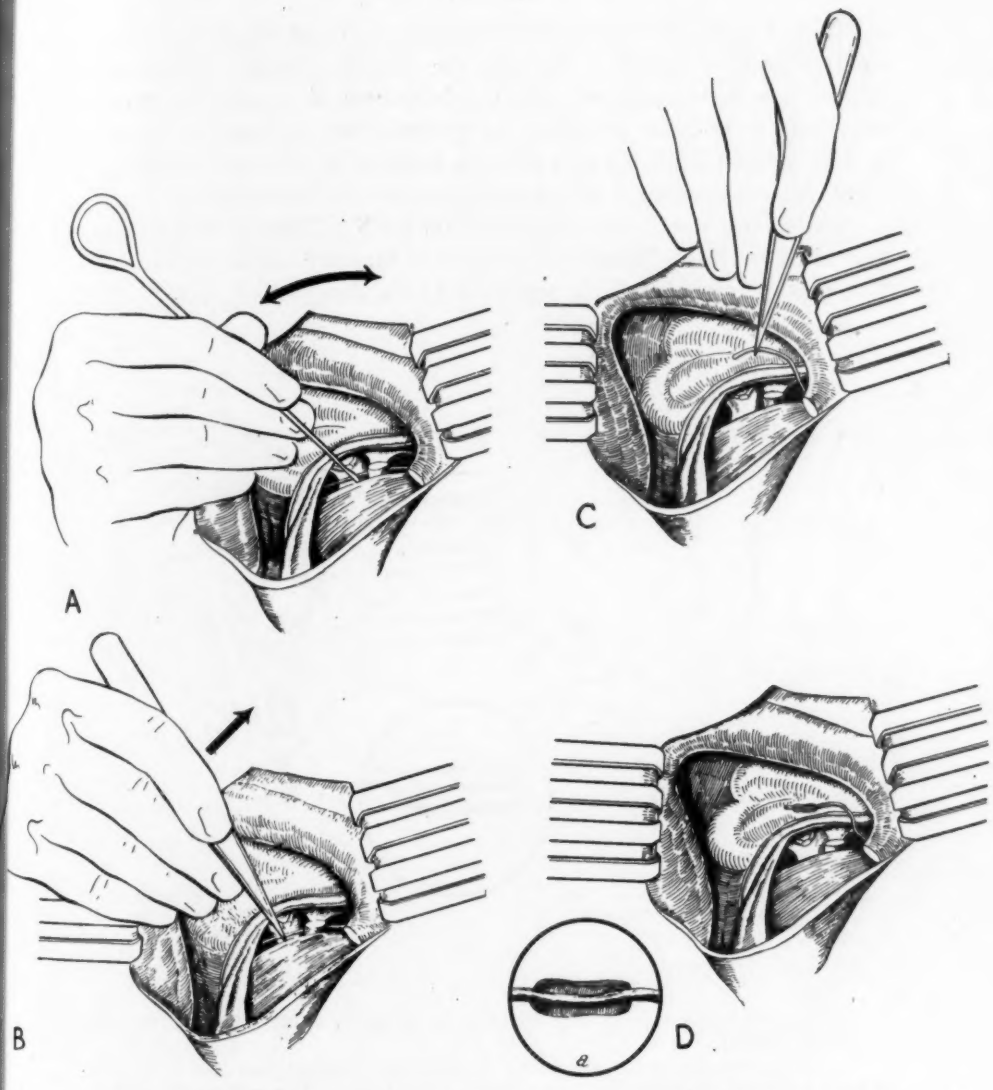


Fig. 1.—In *A* the chorda tympani nerve has been separated from the drum membrane with the blunt edge of a myringotomy knife.

In *B* the nerve is grasped and pulled upward.

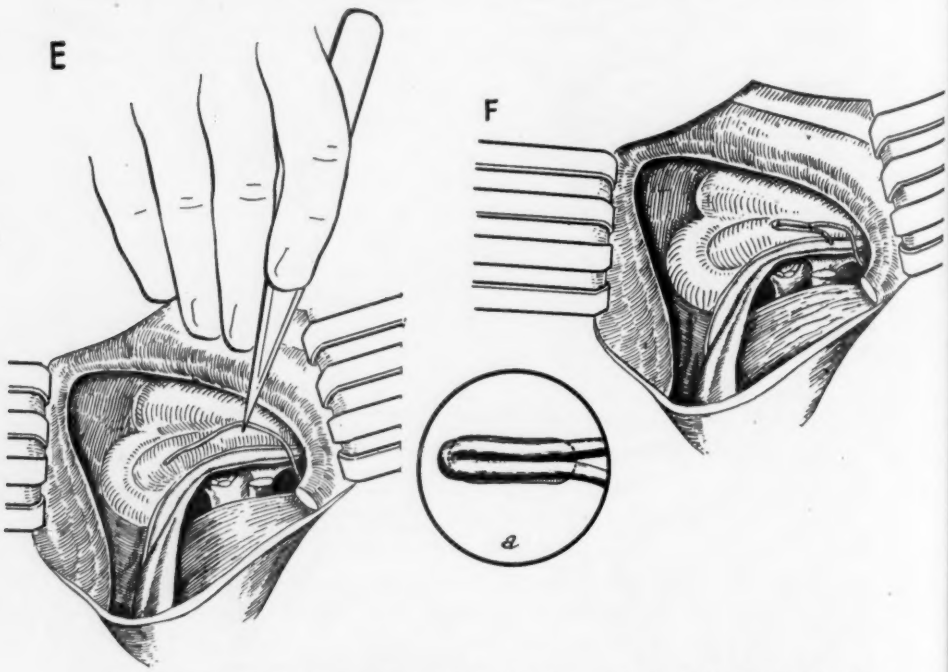
C, the nerve has been pulled out of its bony canal (about two thirds of its length has been delivered).

D, the nerve overlies the membranous labyrinth in the fenestra. It is one half the width of the fenestra. A small amount of perilymphatic space is visible on each side of the chorda (*a*). There must be sufficient slack in the proximal end of the chorda tympani so that when the flap is laid down it will not distort the position of the chorda over the fenestra.

RESULTS

Table 1 gives the audiometric readings at frequency 1024 in 8 consecutive patients operated on with the chorda tympani graft technic. All patients were measured over the full range of frequencies at every examination, but, for simplicity of presentation, audiometric readings in this and the following two tables are given for frequency 1024 only. They are representative of the entire range of frequencies.

Within two weeks after the operation the 8 patients all had a decibel loss of 25 or less. Hearing continued to improve rapidly, and by the fifth postoperative week the majority had a decibel loss of 15 or less.



E, the entire nerve has been pulled out of its canal, after being detached from the facial nerve.

F, evulsion of the entire nerve after it has been detached from the facial nerve allows a double loop to cover the fenestra completely. There must be sufficient slack in the proximal end of the chorda tympani so that when the flap is laid down it will not distort the position of the chorda over the fenestra.

This level of hearing has been maintained or improved to date. It may be seen that in all cases a level of 25 or better has been maintained. Of the 5 patients in whose cases seven months have elapsed since operation, 3 now hear normally (loss 5 decibels or less).

As shown in table 1, significant recovery of hearing is noted in most patients by the end of the second postoperative week. To investigate this finding a survey of the published literature was made for

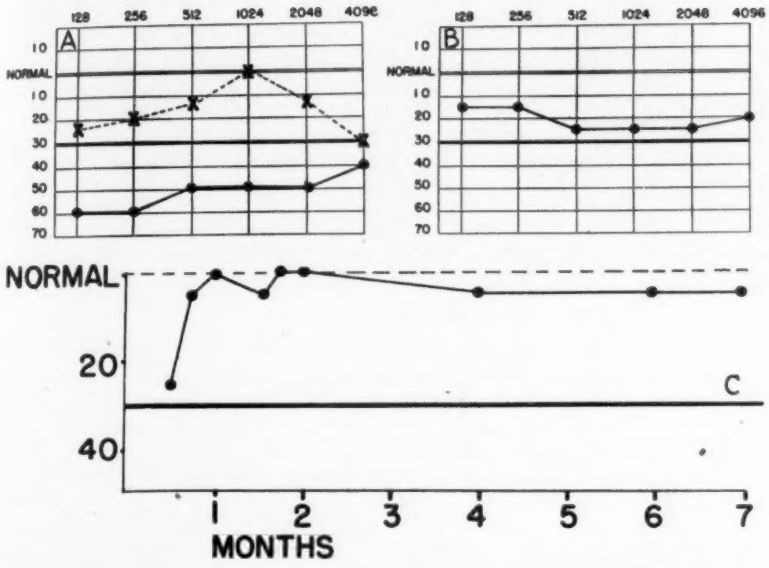


Fig. 2.—Audiograms of F. W. *A*, preoperative; *B*, two weeks postoperative; *C*, from one to seven months postoperative. The test frequency is 1024 cycles per second. The solid line represents the threshold of hearing by air conduction; the broken line, that by bone conduction.

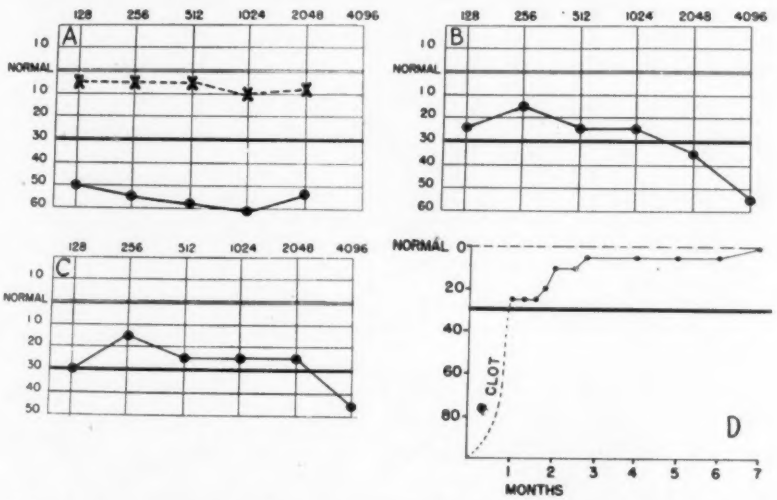


Fig. 3.—Audiograms of N. R. *A*, preoperative; *B*, four weeks postoperative; *C*, six weeks postoperative; *D*, one to seven months postoperative. The test frequency is 1024 cycles per second. The solid line represents the threshold of hearing by air conduction; the broken line, that by bone conduction.

cases in which the hearing had been tested during the early postoperative weeks. Inasmuch as published data on this subject are scanty, I have secured four series of consecutive cases for purposes of comparison. Drs. G. Shambaugh Jr., Howard P. House, W. D. Currier and Louis Z. Fishman each made available results on a consecutive series of cases for purposes of comparison.

Table 2 shows a comparison of the median observations in the four series and in the present series.[5] Week by week comparison shows that the early recovery of useful hearing observed in the present series (table 1) is unusual. During the first six postoperative weeks the hearing of the present series is 5 to 20 decibels better than that

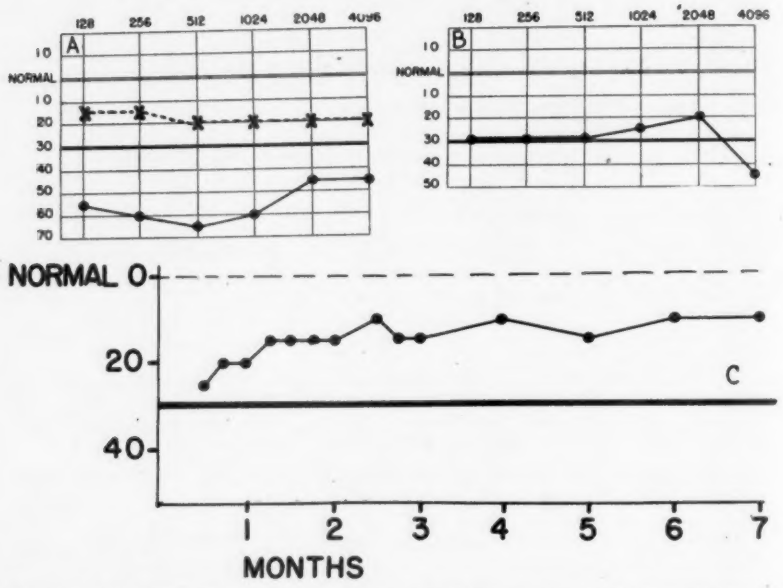


Fig. 4.—Audiograms of M. B. *A*, preoperative; *B*, two weeks postoperative; *C*, from one to seven weeks postoperative. The test frequency is 1024 cycles per second. The solid line represents the threshold of hearing by air conduction; the broken line, that by bone conduction.

obtained by the usual fenestration technics, and it remains so for the first six months.

It is too early to know whether the beneficial results will be maintained. Only time will provide the answer. It has, however, been possible to compare early and year end audiometric records of 57

5. The use of the median avoids the overemphasis of extreme cases which occurs when the average is used. The median is the exact middle of a series (e.g., in a series of 25 values arranged in numerical progression the median is the thirteenth value). In table 2 use of the average would increase the difference between the present series and series A, B, C and D.

patients on whom the fenestration operation was performed. These data are likewise derived from the four series made available to me and are shown in table 3.

Table 3 was made by entering each patient at the point corresponding to his decibel loss for frequency 1024 at six weeks and at one year. The reading of the table is illustrated as follows, reading horizontally: On the second row of entries there were 13 patients whose six week

TABLE 1.—*Audiometric Readings at Frequency 1024 in Eight Cases in Which the Chorda Tympani Nerve Graft Was Used*

Patients	F. W.	N. R	P. S.	R. T.	M. B.	M. L.	B. A.	D. S.	
Preoperative	50	60	50	65	60	45	55	55	
Postoperative									Median
2 weeks	25	Clot	20	25	25	25	25	..	25
3 weeks	5	Clot	25	25	20	15	..	30	25
4 weeks	0	25	15	20	20	15	..	25	20
5 weeks	5	25	15	25	15	10	..	10	15
6 weeks	5	25	15	25	15	..	15	20	15
7 weeks	0	20	15	25	15	15	..	20	15
8 weeks	0	10	15	25	15	10	10	30	13
9 weeks	Otitis	10	15	25	..	..	..	20	13
10 weeks	Otitis	10	15	25	10	10	10	15	10
11 weeks	Otitis	5	15	15	15	..	..	25	15
3 months	Otitis	Head cold	10	15	15	10	15	..	10
4 months	5	5	15	20	10	15	20	..	15
5 months	..	5	25	20	15	..	..	..	15
6 months	..	5	10	25	10	..	..	..	10
7 months	5	0	5	20	10	..	..	..	5

TABLE 2.—*Comparison of Median Decibel Loss at Frequency 1024 in Five Series of Cases of Fenestration*

Series	A	B	C	D	This Series
Patients	20	25	14	26	8
Postoperative reading at:					
2 weeks	..	40	..	45	25
3 weeks	..	..	..	40	25
4 weeks	..	30	20	..	20
6 weeks	30	30	20	30	15
3 months	..	30	..	30	10
6 months	25	30	32	40	10

reading was 20 decibels. At the end of the year 7 of these patients still had 20 decibels, 4 had 25, 1 had 30 and 1 had 40 decibels.

The table can also be read the other way; that is, vertically: In the second row of entries there were 10 patients who had a loss of 20 decibels at the end of the year. Of these patients, 1 had 15 decibels, 7 had 20 decibels and 2 had 25 decibels at six weeks.

While not directly useful in predicting the outcome in the present series, this table has a bearing on two related subjects:

(*a*) To what extent are early postoperative audiometric readings representative of the results obtained at the end of the year?

In the table heavy lines have been drawn corresponding to readings 10 decibels above and 10 decibels below the six week audiometric reading. Inspection of the table shows that only 7 patients fell outside of the lines. The interpretation is that 88 per cent, that is, 50 out of 57 patients at the end of the year had readings within 10 decibels plus or minus of their six week readings. Also, 65 per cent, that is, 38 out of 57 patients, at the end of the year had readings within 5 decibels of their six week readings.

(*b*) Considering only favorable early cases, to what extent is the improvement maintained at the end of the year?

In the table there are a total of 26 patients whose six week tests showed 25 decibels or better (of these 3 had 15, 13 had 20 and 10

TABLE 3.—*Comparison of Six Week and One Year Audiometric Readings at Frequency 1024 in Fifty-Seven Cases of Fenestration*

Vertical rows show number of patients with given decibel loss at one year

Horizontal rows show number of patients with given decibel loss at six months	10	15	20	25	30	35	40	45	50	55	60	65+	Decibels
Patients (total number 57)	1	10	12	7	6	8	6	4	1	1	1		10
3		1	2										15
13		7	4	1		1							20
10		2	2	2	3	1							25
8			2	2	1	2					1		30
3	1		1	1									35
8			1		1	3	2	1					40
4						1	1	2					45
2					1		1						50
4				1			2	1					55
1									1				60
1												1	65+

The table shows that readings at six weeks are indicative of results at one year. Among 57 patients followed for a year, the readings at six weeks were within 5 decibels of the final reading in two-thirds (38 patients) and within 10 decibels in seven-eighths (50 patients).

had 25 decibels). At the end of the year only 8 per cent, 2 among 26, had their reading fall to 40 decibels. As a further point, 69 per cent, 18 patients, had readings of 25 or better.

Thus far osteogenic closure of the fenestra has not occurred in any case. It is too early to predict to what degree, if any, the chorda tympani graft may succeed in preventing osteogenesis.

COMMENT ON RESULTS

Initial results of the use of the chorda tympani nerve graft appear promising: They suggest the possibility of earlier improvement of hearing and achievement of higher average levels of hearing. Noteworthy in this small series are: 3 patients with normal hearing (threshold at 5 decibels or better) at seven months; 1 patient with a

postoperative clot over the fenestra, who nevertheless has normal hearing at seven months; 1 patient with a tear of the flap close to the fenestra whose hearing loss is 10 decibels at seven months.

Definite conclusions must await a longer clinical trial and the accumulation of additional cases. These preliminary results are presented in order to stimulate further investigation in this field.

SUMMARY

A modification of the Lempert fenestra nov-ovalis operation is described.

A series of 8 consecutive patients operated on with the new technic showed recovery of useful hearing within two weeks and continued improvement during the first seven months.

Data are presented from four other series of cases showing the results of early postoperative audiometric testing and the four series and the present series are compared with respect to the six week and one year audiometric results.

101 East Seventy-Third Street.

DIAGNOSIS OF SCLEROMA

MILTON GJELHAUG LEVINE, M.S., Ph.D.
AND
ROBERT E. HOYT, M.S., Ph.D.
LOS ANGELES

ALTHOUGH SCLEROMA is clinically and pathologically a recognized disease entity and many case studies have been published describing the syndrome as seen in the acute form, knowledge of the disease is limited by a lack of precise information concerning the etiologic agent. Chevalier Jackson [1] put the matter mildly when he said, "Though unanimity of opinion is lacking, it is generally accepted that the causative organism is the Bacillus rhinoscleromatis." This acceptance is not in evidence in bacteriologic texts. Gay [2] stated, "K. rhinoscleromatis bears some as yet obscure relationship with the disease rhinoscleroma. It appears, however, commonly to be a secondary invader in the lesions characteristic of the disease." Wilson and Miles [3] said, ". . . there is very little evidence to show that this organism is primarily responsible for it. There is no means by which it can be distinguished with certainty from other members of the capsulated group; and since we know that members of this group may be present in the nose of healthy persons, it is difficult to prove that they play any part in production of rhinoscleroma. The probability is that they are mere secondary invaders which grow freely in the nose of patients suffering from the disease." Zinsser and Bayne-Jones,[4] in discussing the organism, added that there is ". . . further doubt as to its etiological relationship to the disease."

With such frank skepticism among bacteriologists as to the role of the organism in the disease, it is only natural that descriptions of the organism vary.

Part of the difficulty lies in the supposed rarity of the disease, but more in the inability of pathologist, bacteriologist and otolaryn-

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From the Institute of Experimental Medicine, College of Medical Evangelists.

1. Jackson, C., and Jackson, C. L.: Diseases of the Nose, Throat, and Ear, Philadelphia, W. B. Saunders Company, 1945.

2. Gay, F. P.: Agents of Disease and Host Resistance, Springfield, Ill., Charles C Thomas, Publisher, 1935.

3. Topley, W. W. C., and Wilson, G. S.: Principles of Bacteriology and Immunity, ed. 3, revised by G. S. Wilson and A. A. Miles, Baltimore, William Wood & Company, 1946.

4. Zinsser, H., and Bayne-Jones, S.: A Textbook of Bacteriology, New York, D. Appleton-Century Company, Inc., 1939.

gologist to correlate their findings. The disease itself is not too rare. We have come in contact with 16 cases in a relatively short period. If the etiology is clarified, there is no question but that more cases will be recognized. Without the aid of bacteriologic and serologic studies, only typical cases of the acute form are recognized, whereas, as in every other disease, there should occur intermediate and mild, less recognizable stages of scleroma. We have studied the disease extensively, with emphasis on etiologic factors, and, in our opinion, have contributed some clarity to the subject, so that now clinical investigations can be undertaken in order to throw light on the remaining obscure phases of the problem. This paper is intended to enumerate the procedures to be used in the diagnosis of scleroma, and incorporates our own findings with regard to the etiologic agent.

CLINICAL DESCRIPTION

When von Hebra [5] discovered rhinoscleroma in 1870, he assumed it to be an unusual form of cutaneous cancer confined to the nose—hence, the prefix "rhino." Now it is known that the lesions are not limited to the nose but may be found anywhere in the upper respiratory tract, as well as in the external portions of the nose. The more inclusive term, "scleroma," adopted at the Second International Congress of Otorhinolaryngology,[6] held in Madrid in 1932, is much more appropriate.

In its acute form the disease is readily recognized. It is a granulomatous process primarily localized in the upper respiratory tract and secondarily appearing about the piriform aperture of the vestibule of the posterior naris. In the larynx the lesions are usually found in the subglottal space.

In a third of the cases the pharynx is the seat of lesions. These are found in the mesopharynx, occupying the posterior surface of the soft palate, the uvula, the pillars and the lateral portions of the posterior wall. The trachea is occasionally involved. "The apparent involvement of the skin of the upper lip is really a subdermal extension of the submucosal infiltration beneath the anterior nares and the upper lip."[1]

Szmurlo,[7] on the basis of extensive studies in Poland, described secondary lesions of the heart and the lungs

> . . . caused by an obstruction in the respiratory tract, especially in the larynx and trachea, by scleromatous infiltrations which cause a diminution of the intake

5. von Hebra, H.: Ueber ein eigenthümliches Neugebilde an der Nase: Rhinosclerom; nebst histologischem Befunde vom M. Kohn, Wien. med Wchnschr. **20**:1, 1870.

6. Rapports Congrès international d'oto-rhino-laryngologie, Madrid, 1932.

7. Szmurlo, J.: Anatomicopathology of Scleroma, Arch. Otolaryng. **18**:393 (Sept.) 1933.

of air in the lungs, a lowering of intrathoracic pressure, an obstruction of the pulmonary vessels or atelectasis of the alveolus. Hypertrophy of the right side of the heart occurs, and in turn fatty degeneration of the cardiac muscle.

Clinically, the nodular type of lesion described in the foregoing paragraphs is easily recognized. As mentioned previously, a prenodular stage must exist. The lack of specific diagnostic tests prevents its routine recognition. Szmurlo has actually described this form of scleroma as the "diffuse" stage. This first

> . . . appears as atrophic rhinitis characterized by atrophy of the nasal mucosa with abundant secretion and gray crusts on the surface. The atrophic lesions extend along the pharyngeal mucosa, descending to the larynx and trachea, where the characteristic crusts are formed. Besides the atrophic rhinitis there is a diffuse infiltration of the mucosa which extends to the neighboring regions and occupies the subcutaneous tissue, passing to the vestibule of the nose, to the tissue around the pyriform aperture and to the nasopharynx, the lips and the gums. Diffuse infiltration also strikes the pharynx, extending to the mucosa of the soft palate, the uvula and the pillars.

This description is not entirely acceptable to all otorhinolaryngologists, for obvious reasons. It does not have the easily recognizable definition of the nodular type.

PATHOLOGY

The gross lesion most frequently described is the circumscribed, nodular type, which may appear as a single nodule or in groups. The nodules may vary in size from 1 to 10 mm. Their consistency varies; the early infiltrates are soft, while those of longer standing are harder.

Histologically, the picture is that of a granuloma. The ciliated epithelium is transformed into stratified pavement cells. In the deeper layers of the cornea, the protoplasm becomes more dispersed and partially disappears; vacuoles and pyknotic nuclei are seen. Perivascular infiltration is found in which the Mikulicz[8] cells stand out. Szmurlo's own description of the remaining microscopic details of the disease adequately completes the picture:

> Homogeneous globules of different sizes are found, which stain deeply with acid fuchsin. These are Russell's bodies. Besides the Russell bodies there are homogeneous masses in places which stain well with eosin and acid fuchsin. The vessels are numerous and sometimes dilated, and in many there is an increased number of endothelial cells. The length of the vessels of the bundles of connective tissue which penetrate the interior of the cellular conglomeration is next observed. The plasma cells undergo various modifications. The protoplasm gradually melts and disappears, the nuclei become free and undergo karyokinesis, and the destruction of the cells gradually ends. The cells then begin to grow, become homogeneous and undergo hyaline degeneration. They are even transformed into fibroblasts and give rise to connective tissue. Mikulicz's cells are also large and of diverse forms. Large cells with voluminous vacuoles, filiform protoplasm and small pyknotic nuclei are found; some are the size of plasma cells, containing much protoplasm, small vacuoles and one or two nuclei

8. Mikulicz, J.: Ueber das Rhinosclerom, Arch. f. klin. Chir. **20**:485, 1877.

of ordinary volume; some contain diplobacilli which are colored distinctly by hematoxylin or by the Gram stain as modified by Weigert. The diminished parvicellular infiltration is gradually replaced by fibrous connective tissue which hinders the formation of necrotic foci despite a live process of disintegration. The plasma cells and Mikulicz's cells disappear, and only the lymphocytes and rare Russell's bodies are seen. The development of collagenic fibrous connective tissue is most accentuated around the vessels. In some cases there are considerable increase and multiplication of the endothelium; some vessels are thrombosed, and their walls present hyalinization. The same degenerative process may be observed in the connective tissue which is transformed into cicatricial tissue.

BACTERIOLOGY

In 1882 von Frisch[9] described a gram-negative mucoid bacillus as the cause of the disease. Since it was impossible to produce an experimental lesion in animals, the organism was not generally accepted. Bergey's Manual,[10] however, still lists the organism as Klebsiella rhinoscleromatis. Bergey's list of its characteristics is based on the original description and not on later laboratory evidence. Most recent texts merely quote the old literature as does the paper by Perkins,[11] published in 1904, which is obviously inadequate because of the antiquated bacteriologic methods of the period. Publications scattered through the years have added to the confusion with unreliable bacteriologic data reported secondarily to studies of isolated cases. Berge,[12] for example, on the basis of a single case seen in Tunis describes the organism isolated. A rather peculiar statement, however, negates the entire description: *"La culture sur gélose nous a permis d'isoler un bacille Gram négatif associé à un streptocoque."* (The culture on agar permitted us *to isolate a gram-negative bacillus associated with a streptococcus*). It is not quite clear what is meant by the italicized phrase unless the author was dealing with a contaminated culture.

Much of the etiologic difficulty encountered is due to the allegation that the bacillus or scleroma or related organisms are present in normal throats. To test this hypothesis, one must establish a criterion as to the characteristics of the bacillus of scleroma; then one must study the frequency with which it occurs in the normal throat. The very early literature may be discarded, since the required facts about the related Friedländer bacillus and the coli-aerobacter group were not

9. von Frisch, A.: Zur Aetiologie des Rhinoskleroms, Wien. med. Wchnschr. **32**:969, 1882.

10. Bergey, D. H.; Breed, R. S.; Murray, E. G. D., and Hitshens, A. P.: Bergey's Manual of Determinative Bacteriology, Baltimore, Williams & Wilkins Company, 1938.

11. Perkins, R. G.: Bacillus Mucosus Capsulatus: A Study of the Group and an Attempt at Classification of the Varieties Described, J. Infect. Dis. **1**:241, 1904.

12. Berge, C.: Étude bactériologique d'un bacille de Frisch isolé du premier cas de rhinosclérome publié en Tunisie, Arch. Inst. Pasteur de Tunis **31**:197, 1942.

available to the first investigators. There is, however, a remarkable uniformity, which has been overlooked, in the descriptions of the bacillus of rhinoscleroma, published by a number of reliable investigators in recent years. Figi and Thompson,[13] in 1928, described three strains isolated from patients at the Mayo clinic. Morris and Julianelle,[14] apparently unaware of Thompson's report, studied nine strains from cases in Sumatra and this country. Both of these workers found that their strains fermented dextrose, maltose and mannite with the production of acid only. Sucrose was fermented slowly by some strains, lactose never. Indole was not formed. Nitrate was reduced. Other reactions are noted but are not essential to the differential identification of the organism. Gonsiorowski and Meisel,[15] two Polish workers with a large number of freshly isolated strains at their disposal, reported the fermentation of dextrose (acid only) but not of lactose, the only sugars tested.

We have isolated eleven strains from cases to be described later in this paper and have obtained one culture from the American Type Culture Collection. As a result of cultural studies of these organisms we have concluded that the characteristic cultural pattern is as follows:

Lactose	Dextrose	Maltose	Mannite	Sucrose	Indole	Nitrate Reduction
—	Acid	Acid	Acid	Acid or —	—	+

We feel that the characteristics described, including the variable fermentation of sucrose, should be considered specific for these organisms. If we include the colony characteristics (large, translucent, mucoid, coalescing colonies on blood agar, nutrient agar and eosin–methylene blue agar), we have a sufficient differential description to identify this organism.

No other related gram-negative rod found on the human subject has been reported which gives the same pattern of reactions. The variably fermenting Friedländer group may sometimes cause confusion. However, Morris and Julianelle [14] have reported that the two species may be distinguished serologically if occasional strains of the Friedländer bacillus should give a cultural pattern similar to that of the bacillus of rhinoscleroma. There is an interesting antigenic relationship between the latter and the Friedländer group C. Despite a difference in cultural reactions, group C exhibits antigenic similarity when compared serologically with the bacillus of rhinoscleroma.[14] Further work is in progress in our laboratory on this aspect of the etiologic problem.

13. Figi, F. A., and Thompson, L.: Rhinoscleroma, J. A. M. A. **91**:637 (Sept. 1) 1928.

14. Morris, M. D., and Julianelle, L. S.: A Biological Classification of the Bacillus of Rhinoscleroma, J. Infect. Dis. **55**:150, 1934.

15. Gonsiorowski, N., and Meisel, H.: Sur la biologie de Bacilles muqueux encapsules, Compt. rend. Soc. de biol. **100**:298, 1929.

We next tested the assumption that the bacillus of scleroma is found in normal throats. A total of 204 normal persons and 63 with various ailments of nose and throat were studied bacteriologically. The procedure used was found to be satisfactory in the isolation of the organisms of rhinoscleroma in the known cases of infection. Nose and throat swabs representing all the infected persons were streaked on blood agar and eosin–methylene blue agar. Since the results obtained on blood agar and eosin–methylene blue agar were similar, we employed only the latter in our studies of the normal persons and those with nasopharyngeal complaints.

Gram-negative organisms (which morphologically are related to the bacillus of scleroma) occurring in the nose and throat that will grow on eosin–methylene blue agar include members of the coli-aerogenes and Friedländer groups, Proteus, Pseudomonas and Alkaligenes. Most noses and throats did not contain a sufficient number of any of these to give a positive culture. Of a total of 267 persons tested, only 34 had positive cultures. The results of the cultural studies of the organisms isolated are presented elsewhere.[16]

Although we have not as yet separated the isolated strains into species, it is obvious that none has the pattern which we have found to be characteristic of the bacillus of scleroma. In our experimental experience, therefore, this bacillus is not found in the normal nose or throat.

The finding that a specific organism can be isolated in all cases of scleroma and only in such cases certainly indicates more than a chance relationship between the organism and the disease. This finding is even more important when we consider the constancy with which the organism may be demonstrated in the sectioned granulomatous tissue. Finally, if we include the serologic findings to be discussed in the next section, we cannot help assuming that the organism is associated with the disease.

SEROLOGY

As is true of the cultural studies, the early reports of serologic studies are confusing, and for the same reasons. However, more recent studies, if correlated, certainly are not to be disregarded. Goldzieber and Neuber,[17] Brault and Masselot[18] and Quast[19] have all agreed

16. Levine, M. G.; Hoyt, R. E., and Peterson, J. E.: Scleroma: An Etiological Study, J. Clin. Investigation **25**:281-286, 1947.

17. Goldzieber, M., and Neuber, E.: Untersuchungen über das Rhinosklerom, Centralbl. f. Bakt. **51**:121, 1909.

18. Brault, J., and Masselot, L.: Quelques remarques sur le bacille du rhinosclérome (bacille de Frisch), Arch. de méd. expér. et d'anat. path. **22**:545, 1910.

19. Cited by Mayer, E.: Scleroma of the Larynx, Am. J. M. Sc. **133**:751, 1907.

that the serums of patients with scleroma or of rabbits immunized with the organism are capable of fixing complement. Fitzgerald [20] showed that a rabbit immune serum agglutinated specifically the decapsulated organism. Prašek and Prica [21] and Morris and Julianelle [14] have both demonstrated the specific antigenicity of the organism.

We have previously reported successful results with the complement fixation test.[16] Our most recent experience is even more gratifying. Using a heat-killed (65 C. for thirty minutes) antigen, we tested twelve serums [22] from patients known to have scleroma. All showed marked positive reactions, whereas only 15 of 534 serums from patients with other assorted diseases gave positive reactions, none of these as strongly positive as those given by the serums from patients known to have scleroma.

The details of the complement fixation tests are reported elsewhere.[23] Certainly we may conclude that the complement fixation test is a valuable aid in the diagnosis of scleroma.

EPIDEMIOLOGY

Scleroma was once thought to be confined to Central and Southeastern Europe, but it is now admitted that the disease is worldwide in distribution. Cunning and Guerry [24] reported in 1942 that 102 cases had been recorded in the American and Canadian literature to that date. Streit [25] and others expressed the opinion that an endemic area exists in Central Europe, especially in Poland. Lasagna [26] stated that it is being recognized more frequently in Italy. In our own experience, in California, the disease has been found in people of Mexican extraction. One family of 6 and 2 unrelated persons of the Los Angeles area have been of Mexican extraction. A series of 3 California cases reported by Alderson [27] concerned 1 Mexican, 1 person from San Salvador and 1 native American. We are, however, receiving specimens

20. Fitzgerald, J. G.: A Biometrical Study of the Mucous Capsulatus Group, J. Infect. Dis. **15**:268, 1914.

21. Prašek, E., and Prica, M.: Agglutinationsreaktion bei Rhinosclerom, Centralbl. f. Bakt. (Abt. I) **108**:376, 1928.

22. One serum was obtained from Dr. M. Gerundo, of Hilo, Hawaii, and four from Dr. M. Ruiz Castaneda, of Mexico City.

23. Levine, M. G., and Hoyt, R. E.: Complement Fixation Test in Scleroma (Rhinoscleroma), Proc. Soc. Exper. Biol. & Med. **65**:70-72, 1947.

24. Cunning, D. S., and Guerry, D.: Scleroma, Arch. Otolaryng. **36**:662 (Nov.) 1942.

25. Streit, H.: Etiology of Scleroma, Arch. Otolaryng. **18**:392 (Sept.) 1933.

26. Lasagna, F.: Infective Scleroma of the Respiratory Passages, J. Laryng. & Otol. **40**:369, 1925.

27. Alderson, H. E.: Scleroma in California, California & West. Med. **47**: 317, 1937.

from other parts of the country, and epidemiologic conclusions cannot be drawn until many more cases are studied.

Of significance in studies of contagion is the duplication of cases in the same family, instances of which have been reported by Lasagna,[26] Robertson and Secretan,[19] Gerber [28] and ourselves.[16] Our own series is extremely interesting, since we discovered 6 cases in one family.

The family consists of a father, four daughters and two sons. All, including the father, are native born, of Mexican extraction. The father, aged 37, has had the disease for ten years. The older son, aged 20, has had the disease for about one year. The daughters, aged 19, 17, 14 and 11, were patients with unrecognized scleroma until studied by us. A brief description of these cases has been reported.[16] A more complete rhinologic study is in preparation.

Because of the slow progress of the disease, problems of contagion are difficult to solve. Our own impression is that the epidemiology may be similar to that of leprosy, long exposure to which is essential for infection.

THERAPY

Recent studies on antibiotic agents relate the therapy to the etiology of infectious conditions. Until now the therapy of scleroma has consisted of high voltage roentgen irradiation and surgical removal. Now the knowledge that streptomycin acts against gram-negative organisms offers a suggestion as to therapy. Because of the high cost of streptomycin, our experiments in human subjects have been limited, but, by following cultures of material obtained from the throats, we have observed a marked effect on the bacillus of scleroma when the drug is given. Miller [29] has observed a seeming cure of advanced scleroma with streptomycin. In vitro the organism is not affected by penicillin, slightly inhibited by the sulfonamide compounds and completely inhibited by 1 mg. of streptomycin per hundred cubic centimeters of yeast beef broth. Further clinical trials are certainly in order.

SUMMARY

A discussion of methods by which scleroma may be diagnosed is presented, with emphasis on the etiologic agent. Chemotherapy is suggested as a means of treatment.

312 North Boyle Avenue.

28. Gerber, P. H.: Ueber das Sklerom, insbesondere in Ostpreussen, Arch. f. Laryng. u. Rhin. **10**:347, 1900.

29. Miller, H. A., read before the Los Angeles County Medical Society, Otolaryngological Section Meeting, Feb. 24, 1947.

HEMORRHAGE IN DEEP INFECTIONS OF THE NECK

A Review, with Report of Two Cases

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INFECTIVE and suppurative diseases of the neck are nearly always secondary to primary foci in the head. Infections of tonsils, pharynx, teeth and nasal accessory sinuses are the most common etiologic sources. Otitis media, a Bezold abscess in mastoiditis, petrositis, infection of the base of the tongue, infection of the floor of the mouth, thrombosis of the pterygoid plexus of veins—all have been responsible for deep infections of the neck. Infected lymph glands, especially those in the retropharyngeal space, may be the source of a deep cervical abscess. Trauma occurring in the mouth, in the pharynx or the esophagus as a result of the presence of a foreign body is occasionally causative. A tuberculous abscess of the body of one of the cervical vertebrae may rupture and burrow into a deep fascial plane of the neck to give rise to a cold abscess.

Infection may be carried from one of the aforementioned sites by one of three routes: (1) the blood stream; (2) the lymph channels; (3) direct implantation. An example of the last would be a needle with procaine hydrochloride passing through an infected tonsil into the pharyngomaxillary space during the performance of tonsillectomy.

Pathologically, deep infections of the neck are of two types—phlegmonous and vascular. Both varieties may be present. Most common is the phlegmon, which begins as cellulitis in the loose areolar tissue of a deep cervical fascial plane and develops, if not arrested, into an abscess with the latter's signs and symptoms. Occasionally, the vascular type is seen. This presents an appearance of septicemia and, perhaps, thrombosis and embolism with little evidence of the origin of the infection. Thrombosis of the internal jugular vein is the outstanding example of this type. According to Mosher,[1] thrombosis of the internal jugular vein

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From the Otolaryngologic Service, Morrisania City Hospital.

Read before the Bronx Otolaryngological Society, Nov. 26, 1946.

1. Mosher, H. P.: Submaxillary Fossa Approach to Deep Pus in Neck, Tr. Am. Acad. Ophth. **34**:19, 1929.

is occasionally the cause of a type of septicemia which ends fatally without an etiologic diagnosis. It generally originates from a tonsillar infection.

In the course of phlegmon of the neck, the large vessels of the neck may become infected. Veins are infected more often than arteries; the internal jugular vein is the most vulnerable. Occasionally the internal carotid artery or one of the branches of the external carotid artery becomes involved. It is important to note that phlebitis develops into thrombosis and that arteritis leads to erosion of the vessel wall without the formation of a thrombus. When an artery is caught in an abscess, erosion of the arterial wall leads to profuse and often fatal hemorrhage. This paper will present 2 cases of fatal hemorrhage resulting from erosion of the wall of an artery in the course of deep suppuration of the neck.

SURGICAL ANATOMY

From a surgical standpoint the deep cervical fascia is the most important structure in the neck. According to Barnhill,[2] it is the key to a proper understanding of the pathways of infection and is an aid in diagnosis and rational surgical treatment. The superficial layer of the deep cervical fascia is a continuous fibrous sheath which completely envelops all the structures of the neck from the mandible and the occiput above to the sternum and the clavicle below. It is because of this unyielding covering that pus in the neck burrows deeply along the fascial planes and does not point subcutaneously.

The so-called splitting layer of the deep cervical fascia encloses the trapezius muscle behind, the sternocleidomastoid muscle laterally and the subhyoid muscles in the front of the neck. It is attached to the hyoid bone and from there extends upward and splits to enclose first the submaxillary gland and then the parotid gland. The deep layer under the parotid gland thickens into the stylomandibular ligament, which runs from the styloid process to the inner surface of the angle of the mandible and separates the submaxillary from the parotid gland.

The fascia under the sternocleidomastoid muscle gives off the alar process which splits to form the carotid sheath. At the innermost part of the carotid sheath the two layers unite again to form the prevertebral layer of fascia, which encircles the vertebral column and the muscles attached to it. The visceral fascia encircles the pharynx and the larynx above and the trachea and the esophagus below in a single compartment. In the upper part of the neck the visceral fascia is known as the buccopharyngeal fascia and encloses all the constrictor muscles of the pharynx.

2. Barnhill, J. F.: Surgical Anatomy of the Head and Neck, ed 1, Baltimore, William Wood & Company, 1937.

Between these layers of fascia are potential spaces which become actual spaces when distended with pus. One such area is found in the floor of the mouth; a second is deep to the submaxillary and parotid glands; a third is found in the retropharyngeal area, and, finally, a fourth is seen along the carotid sheath. It is rare to find a primary deep abscess of the neck in any other than one of these four locations.

The retropharyngeal space is situated in the posterior part of the pharynx and lies between the buccopharyngeal fascia covering the constrictors of the pharynx in front and the prevertebral fascia which covers the vertebral column and the muscles attached to it behind. It extends from the base of the skull above to the posterior mediastinum below. It contains six to eight glands in infancy, which atrophy in the adult. This space is separated from the parapharyngeal space and the carotid sheath laterally by a thin layer of deep cervical fascia, which when pierced allows pus from the retropharyngeal space to invade the pharyngomaxillary space and in turn the carotid space.

The pharyngomaxillary space is a large triangular compartment located lateral to the pharynx. Its base is at the base of the skull around the foramen of the jugular vein, and its apex is at the hyoid bone. It is separated by the styloid process into a prestyloid and a poststyloid compartment. The anterior or prestyloid space is only a potential one unless distended by infection. It contains fat, connective tissue, a few lymph glands, the ascending pharyngeal artery and occasionally the external maxillary artery. The posterior or retrostyloid space contains the carotid sheath with the internal carotid artery, the internal jugular vein and the vagus nerve.

The medial boundary of the pharyngomaxillary space is the superior constrictor muscle of the pharynx with the palatine tonsil attached to it. Its lateral boundary below is the internal pterygoid muscle lining the inner surface of the ramus of the mandible, and its lateral boundary above is the inner surface of the parotid gland, which at this point has no fascial covering. Posteriorly the prevertebral fascia and muscles bound the fossa.

Infection of the floor of the mouth is prevented from extending down in the anterior part of the neck by the mylohyoid muscle and then by the resistance of the superficial layer of the deep cervical fascia. Hence pus when it breaks through in this area burrows along the posterior belly of the digastric muscle to its posterior end and pours over into the carotid space. Thus an abscess of the floor of the mouth, the retropharyngeal space or the pharyngomaxillary space tends to invade the carotid space unless drained in time at its source. Once the infection has invaded the carotid space, the purulent exudate, if unimpeded, can burrow along the fascial planes either up toward the base of the skull or down into the anterior mediastinum of the thorax.

TREATMENT OF DEEP INFECTIONS OF THE NECK

The use of chemotherapeutic drugs, penicillin and streptomycin in adequate doses should abort the spread of most deep infections of the neck. In the early stages, when a localized abscess has developed, a simple intraoral incision will suffice. This should be made in the floor of the mouth, in the posterior pharyngeal wall or in the lateral pharyngeal wall just behind the posterior pillar of the tonsil, depending on whether the infection is Ludwig's angina (cellulitis of the floor of the mouth), a retropharyngeal abscess or a parapharyngeal abscess, respectively. It is important, before making the incision, to aspirate the swollen area to make sure that pus and not blood is present. An aneurysm or an eroded artery will thus not be accidentally incised.

When the infection has gone beyond the bounds of the original space involved or when there has been no tendency for any localization to occur, an approach in the neck becomes necessary. In Ludwig's angina localization is the exception rather than the rule and early incision of the neck is the treatment of choice. Mosher [1] has devised a submaxillary approach to deep cervical abscesses. He makes an incision parallel and close to the lower border of the mandible. The submaxillary gland is lifted out of its bed. The finger is inserted beneath the gland and pushed backward and upward until the stylomandibular ligament is felt. The finger passes along this ligament until the styloid process is palpated. The pharyngomaxillary space is entered by inserting the finger upward and external to the styloid process. With pus in the floor of the mouth or at the base of the tongue, the same external approach is made, followed by an incision of the center of the bed of the submaxillary gland. The probing finger is inserted into this opening and carried forward or backward to locate the pus, according to the indications.

Beck [3] uses a single incision running parallel to the border of the mandible. Instead of displacing the submaxillary gland, he exposes the deep fascia at a point behind the angle of the mandible. A closed blunt arterial forceps is inserted through the deep fascia at the angle of the jaw, and the finger is introduced through this opening. The stylomandibular ligament and the styloid process are identified, and the space is entered. The carotid sheath lies behind the styloid process and may be drained at this point if necessary.

The carotid sheath may also be drained by exposing this space through an incision along the upper half of the anterior border of the sternocleidomastoid muscle. It is by this approach that the carotid arteries (common, internal and external) and the internal jugular vein

3. Beck, A. L., cited by Ballenger, W. L., and Ballenger, H. C.: Nose, Throat and Ear, ed. 7, Philadelphia, Lea & Febiger, 1938, chap. 22.

are exposed and ligated when these vessels are involved in deep infections of the neck.

REPORT OF CASES

CASE 1.—R. H., a white boy aged 4 years, was admitted to the Morrisania City Hospital to the otolaryngologic service of Dr. Goodlatte B. Gilmore on April 12, 1939 with a hemorrhage of the throat. The illness began two weeks previously with a soreness of the throat. A week later the child had a nasal "twang" to his voice and it was observed that he had a swollen gland in his neck at the angle of the left jaw. On the day of admission, after an incision had been made in the throat by his private physician, he had a profuse hemorrhage of the throat and respiratory difficulty. He was taken to the hospital immediately. On admission he looked critically ill. He was pale and dyspneic, with a temperature of 104.6 F., a pulse rate of 164 and respiratory rate of 42. His skin was cold and clammy.

Examination revealed marked swelling about the left tonsil and an incision in the soft palate above the left tonsil from which neither blood nor pus was coming. There was swelling about the left parotid gland and in front of the left ear. A small amount of blood was noted in the canal of the left ear but the drum was normal. A diagnosis of abscess of the left pharyngomaxillary space with erosion of a large artery, causing hemorrhage, was made.

A transfusion of 250 cc. of whole blood, followed by a continuous infusion of 5 per cent dextrose in solution of sodium chloride was given on admission. The child's general condition improved rapidly following the transfusion. No bleeding was seen in the throat. There was no bleeding during the night or on the following morning. In view of the improved condition of the patient and the absence of bleeding, supportive measures and expectant treatment were recommended. At about noon of the day following the day of admission the patient suddenly began to expectorate frothy bloody fluid, and death followed in a few minutes, the patient apparently drowning in his own blood, from an extensive hemorrhage.

Relevant postmortem observations were as follows: The lungs on section showed aspirated blood; the stomach and intestine contained a large amount of fluid and clotted blood; the other abdominal organs were pale and anemic looking. In the neck, a large mass of edematous glands was found beneath the upper third of the left sternocleidomastoid muscle. The common carotid artery at its bifurcation and the internal and external carotid arteries at this point were intact. The internal and external jugular veins were intact. There was a large necrotic abscess cavity in the left tonsillar fossa, containing clotted blood. The left side of the pharynx was edematous. The autopsy was done by the city medical examiner, and unfortunately he did not determine the specific blood vessel involved.

CASE 2.—R. H., a white boy aged 7½ years, was admitted to the Morrisania City Hospital to the otolaryngologic service of Dr. Goodlatte B. Gilmore, April 16, 1940, with a hemorrhage of the throat following the making of an incision in the peritonsillar region by his private physician. There was elicited a history of pneumonia with a thoracotomy for empyema at the age of 8 months and frequent episodes of "ear trouble."

The present illness began two weeks previous to admission, with pain in the right ear, for which myringotomy was done. For a week previous to admission the patient experienced increasing difficulty in swallowing. On the day before admission he bled spontaneously from the throat. His physician was called in,

and he incised the left side of the peritonsillar area. As the incision was made, the patient began to bleed profusely, and the hemorrhage continued for fifteen minutes. This bleeding ceased, but on the following day, on the advice of his physician, he was sent to the hospital.

At the time of admission there was no appparent bleeding from the throat. The child did not appear to be acutely ill. At the angle of the left mandible there was a swelling which extended down the neck along the carotid sheath. This area was tender. In the throat there was a diffuse swelling of the left lateral pharyngeal wall. A small incision of the soft palate over the left tonsil did not reveal pus or blood. There were some streaks of blood on the posterior pharyngeal wall. The left drum was normal. The right ear was discharging a scant amount of purulent exudate, and the drum membrane was gray and flat. His temperature was 99.8 F.; the pulse rate was 120 and the respiratory rate 20. The white blood cell count was 44,400, with 94 per cent polymorphonuclear leukocytes and 6 per cent lymphocytes; the red cell count was 3,280,000, with hemoglobin content 75 per cent. A diagnosis of abscess of the left pharyngomaxillary space with erosion of a large artery and subsiding purulent otitis media of the right side was made.

The patient was given a transfusion of 300 cc. of whole blood on admission. For three days he appeared to be getting progressively better. The swelling of the lateral pharyngeal wall diminished, and there was no evidence of bleeding. Blood taken on the third day showed: white blood cells, 17,650, with 84 per cent polymorphonuclear leukocytes and 16 per cent lymphocytes; red blood cells, 4,140,000, with hemoglobin content 80 per cent. Early in the morning of the fourth day, as the child awoke, about 350 cc. of blood gushed from his mouth. He was immediately taken to the operating room.

After induction of general anesthesia, an incision was made along the anterior border of the sternocleidomastoid muscle from an inch (2.5 cm.) below the tip of the mastoid process down to the medial end of the clavicle. The anterior border of the sternocleidomastoid muscle was reflected posteriorly, and by means of blunt dissection the carotid sheath was identified and opened up. The internal jugular vein and the common, internal and external carotid arteries were dissected free from the surrounding tissues. The external carotid artery was ligated above the level of the superior thyroid artery. A no. 12 black silk ligature was placed around the common carotid artery but was not tied. The ends were left outside the wound, to be tied if bleeding occurred later. A piece of rubber tissue was inserted into the carotid sheath, and the wound was partially closed with skin clips. Free pus was found within the carotid sheath at the level of the hyoid bone, and the area surrounding the sheath was infiltrated with dense inflammatory tissue. The patient was given a transfusion of 500 cc. of whole blood, followed by 500 cc. of 5 per cent dextrose in solution of sodium chloride. A nurse was kept at his bedside continuously to watch for bleeding.

The patient improved steadily for three days. On the third postoperative day the blood showed: white blood cells, 32,300, with 84 per cent polymorphonuclear leukocytes and 16 per cent lymphocytes; red blood cells, 2,910,000, with hemoglobin content 50 per cent. On the morning of the fourth postoperative day the ligature around the common carotid artery was removed. There had been no bleeding for four days. The swelling of the throat was subsiding, and the patient appeared better. Suddenly, in the early evening of the fourth day, the patient sat up in bed and began to bleed from mouth and nose. A nurse and then an intern applied pressure over the left carotid artery in the neck, but the child continued to bleed profusely and apparently was drowning in his own blood. Within a few minutes he succumbed.

Blood cultured on the day of admission grew no organisms. Material taken from the carotid sheath at the operation and cultured grew Staphylococcus albus. Throughout the child's stay in the hospital his temperature fluctuated between 99 and 102.8 F. No autopsy was performed.

COMMENT

These 2 cases presented the rather uncommon but certainly most dangerous complication of deep cervical infection—namely, hemorrhage. Profuse bleeding in the course of a suppurative process in deep abscess of the neck means an erosion of an artery, and active measures must be undertaken to obviate a second hemorrhage. The vessel most frequently involved is the internal carotid artery, which lies in the poststyloid compartment of the pharyngomaxillary space. The ascending pharyngeal artery runs through the prestyloid space on its way to the tonsil and is vulnerable in this area. Occasionally the external maxillary artery before it gets to the face loops under the angle of the mandible, and at this point it may become eroded.

In operating in this type of case a problem arises as to the vessel that should be ligated—the external, the internal or the common carotid artery. Ligation of the internal carotid artery is not done without a great deal of misgiving on the part of the surgeon. It causes contralateral hemiplegia in not less than 25 per cent of all cases.[4] This may be transient or permanent. In 10 per cent of all cases, massive softening of the hemisphere occurs, resulting in death. This occurs more frequently in patients over 35 years of age but may occur in young persons in whom the circle of Willis is incomplete. The variability in response to ligation of the internal carotid artery is due to the presence or the absence of collateral circulation.

Matas[5] devised a test to determine before ligation whether the collateral circulation in the circle of Willis would be adequate. He found that if the internal carotid artery could be compressed in the neck for ten minutes without symptoms developing, such as dizziness, numbness or weakness of the contralateral arm, or loss of vision, the artery might be ligated with safety. This test never failed. In 1940 he[6] reported a series of 66 cases in which he had ligated the internal carotid artery for all causes, with eight deaths—a mortality of 12 per cent. Dandy[7] ligated the internal carotid artery in 36 cases, with only 1 death.

4. Ford, F. R.: Diseases of the Nervous System in Childhood, ed. 2, Springfield, Ill., Charles C Thomas, Publisher, 1946, p. 168.

5. Matas, R.: Testing the Efficiency of the Collateral Circulation, J.A.M.A. **63**:1441 (Oct. 24) 1914.

6. Matas, R.: Classified Summary of Six Hundred and Twenty Operations upon the Blood Vessels Performed for All Causes, Tr. Am. S. A. **58**:335, 1940.

7. Dandy, W. E.: Results Following the Ligation of the Internal Carotid Artery, Arch. Surg. **45**:521 (Oct.) 1942.

In our case 1 the patient came in with a history of a hemorrhage, but except for some blood in the external canal there was no sign of active bleeding. He improved so rapidly after the transfusion that nothing further was done. On the day following admission there was no evidence of bleeding and the swelling in the pharynx appeared to be subsiding. Suddenly, without any warning, the patient had a fatal hemorrhage. It was felt that prompt ligation of the internal or external carotid artery or both should have been the treatment in this case.

When the second patient was admitted to the hospital with a history that paralleled that of the first patient, the question of immediate ligation of the carotid arteries arose. The patient, however, showed no evidence of bleeding from the site of the infection in the lateral part of the pharynx. He did not even look acutely ill. When, three days after admission, the patient started to bleed, he was immediately operated on. A ligature placed on the external carotid artery above the superior thyroid artery appeared to stop the bleeding. However, because it was realized that the most common site of bleeding in an abscess of the pharyngomaxillary space is the internal carotid artery, a ligature was placed around the common carotid artery but was left untied. The fact that the patient had a fatal hemorrhage a few hours after this untied ligature was removed was indeed unfortunate.

Lifshutz,[8] in a review of 23 cases of erosion of one of the carotid arteries, usually the internal, reported deaths in 19. Of the 4 patients who survived, 3 had prompt ligation of the common carotid artery. Mosher [1] expressed the opinion that ligation of the common carotid artery does not have the high mortality attributed to it and that it is reasonably safe. Shambaugh [9] expressed the belief that a sudden hemorrhage referable to a deep infection of the neck means an erosion of the internal carotid artery. He stated that while the patient usually survives the first hemorrhage, the second or the third hemorrhage will very likely be fatal. He therefore advocated prompt ligation of the common carotid artery on the side of the abscess to prevent recurrent and probably fatal hemorrhage.

In the presence of a deep infection of the neck hemorrhage requires heroic measures. Early ligation is the conservative rather than the radical procedure. If time permits, the Matas test should be done to determine the adequacy of the collateral circulation in the circle of Willis. Both the internal carotid and the external carotid artery must be ligated above the level of the superior thyroid artery if the patient is to be saved. As an alternate procedure, the common carotid artery may be ligated, but in that event the superior thyroid artery must also be ligated

8. Lifshutz, cited by Barnhill.[2]

9. Shambaugh, G. E., Jr., in Jackson, C., and Jackson, C. L.: Diseases of the Nose, Throat and Ear, Philadelphia, W. B. Saunders Company, 1945.

because its collateral circulation is intimately connected with the inferior thyroid artery on the same side and the superior thyroid artery on the opposite side.

SUMMARY

The causes, the pathologic aspects and the treatment of deep infections of the neck are reviewed.

The significant surgical anatomy of the deep cervical fascia is described.

Two cases of fatal hemorrhage in the course of abscess of the pharyngomaxillary space are reported.

The dangers and the results of ligation of the internal and common carotid arteries are summarized from the literature.

The conclusion drawn from the 2 cases reported and from a review of the literature is that ligation of both the internal and the external carotid artery should be done as soon as a diagnosis of an eroded artery in deep infection of the neck is made. This is the only logical procedure if the patient is to be saved. To hesitate may and usually does mean death from hemorrhage..

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ACUTE LARYNGOTRACHEOBRONCHITIS

Further Studies on Treatment

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DURING recent years many excellent articles describing the treatment of this disease have appeared in the literature. Notable contributions have been made by Richards,[1] Jackson and Jackson,[2] Holinger,[3] Brennemann and associates,[4] Galloway,[5] Font,[6] Brighton,[7] Orton and associates,[8] LeJeune and Bayou,[9] Graebner,[10] McCaskey,[11] Gittins,[12]

From the Department of Otolaryngology and Broncho-Esophagology, George F. Geisinger Memorial Hospital.

Presented before the Eastern Section of the American Laryngological, Rhinological and Otological Society, in Philadelphia, Jan. 13, 1947.

1. Richards, L.: (*a*) Fulminating Laryngo-Tracheo-Bronchitis, Ann. Otol., Rhin. & Laryng. **42**:1014 (Dec.) 1933; (*b*) Further Study of Pathology of Acute Laryngo-Tracheo-Bronchitis in Children, ibid. **47**:326 (June) 1938.

2. Jackson, C., and Jackson, C. L.: Acute Laryngotracheobronchitis: Living Pathologic Conditions Seen in Acute Respiratory Diseases, J. A. M. A. **107**: 929 (Sept. 19) 1936.

3. Holinger, P. H.: Acute Laryngotracheobronchitis, J. Michigan M. Soc. **36**:969 (Dec.) 1937.

4. Brennemann, J.; Clifton, W. M.; Frank, A., and Holinger, P. H.: Acute Laryngotracheobronchitis, Am. J. Dis. Child. **55**:667 (April) 1938.

5. Galloway, T. C.: Postural Treatment of Acute Laryngotracheobronchitis, J. A. M. A. **112**:1566 (April 22) 1939.

6. Font, J. H., and Ortiz, A.: Acute Infective Laryngotracheobronchitis in Puerto Rico, Puerto Rico Health Bull. **3**: 109 (April) 1939.

7. Brighton, G. R.: Laryngotracheobronchitis, Ann. Otol., Rhin. & Laryng. **49**:1070 (Dec.) 1940.

8. Orton, H. B.; Smith, E. L.; Bell, H. O., and Ford, R. A.: Acute Laryngotracheobronchitis, Arch. Otolaryng. **33**:926 (June) 1941.

9. LeJeune, F. E., and Bayou, P. J.: Acute Laryngotracheobronchitis, J. A. M. A. **117**: 1316 (Oct. 18) 1941.

10. Graebner, H.: Chemotherapy in Acute Non-Diphtheritic Laryngotracheobronchitis, Arch. Otolaryng. **35**: 523 (April) 1942.

11. McCaskey, C. H.: Upper Respiratory Infection of a Fulminating Character Requiring Tracheotomy, Ann. Otol., Rhin. & Laryng. **51**:389 (June) 1942.

12. Gittins, T. R.: Laryngotracheobronchitis in Children, Arch. Otolaryng. **36**:491 (Oct.) 1942.

Neffson,[13] Walsh,[14] MacCready,[15] Baum [16] and others. There is still lack of agreement regarding the best methods of treatment. Ten years ago the mortality of this disease was said to be 70 per cent.[2] Since that time succeeding articles have reported gradually decreasing mortality rates. During the past five years I have treated 52 consecutive patients with this disease. There were no deaths in this series, which included 15 patients who required tracheotomy. My purpose in presenting this paper is to analyze the methods of treatment which have made possible this reduction in mortality rate.

CLINICAL PICTURE

Patients in this series all had severe progressive laryngeal obstruction and were sent to the hospital because the pediatrician or the family physician had found that the symptoms failed to respond to treatment.

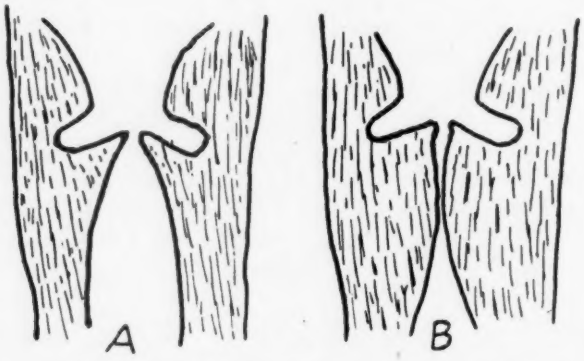

Fig. 1.—*A*, coronal section of a normal larynx; *B*, coronal section of a larynx involved in acute laryngotracheobronchitis, showing the subglottic swelling which causes the obstruction and the croupy cough.

The children had the dry, croupy cough and inspiratory stridor produced by the subglottic swelling indicated in figure 1. All had the retraction of the soft tissues around the clavicles and in the epigastrium during inspiration which is characteristic of any type of laryngeal obstruction. In almost all cases the cry was clear, indicating that the

13. Neffson, A. H.: Tracheotomy in Cases of Acute Obstructive Non-Diphtheritic Infections of the Larynx, Trachea and Bronchi, Arch. Otolaryng. **36**:773 (Dec.) 1942; Tension Pneumothorax and Mediastinal Emphysema After Tracheotomy, ibid. **37**:23 (Jan.) 1943.

14. Walsh, T. E.: Treatment of Acute Laryngotracheobronchitis, Tr. Am. Laryng., Rhin. & Otol. Soc., 1944, p. 222.

15. MacCready, P. B.: On Changing Conceptions of Acute Laryngotracheobronchitis, Ann. Otol., Rhin. & Laryng. **53**:65 (March) 1944.

16. Baum, H. L.: Treatment of Acute Laryngotracheobronchitis, Arch. Otolaryng. **41**:175 (March) 1945.

edges of the vocal cords were normal and free from membrane. At the time of admission the temperature varied from 100 to 105 F. Many patients had little fever. The dyspnea had become progressively worse over a period of one to seven days prior to admission. Direct laryngoscopy was used to differentiate this disease from diphtheria and supraglottic edematous laryngitis. Diphtheria was also ruled out by smears and cultures. If dyspnea is due to laryngeal diphtheria, the

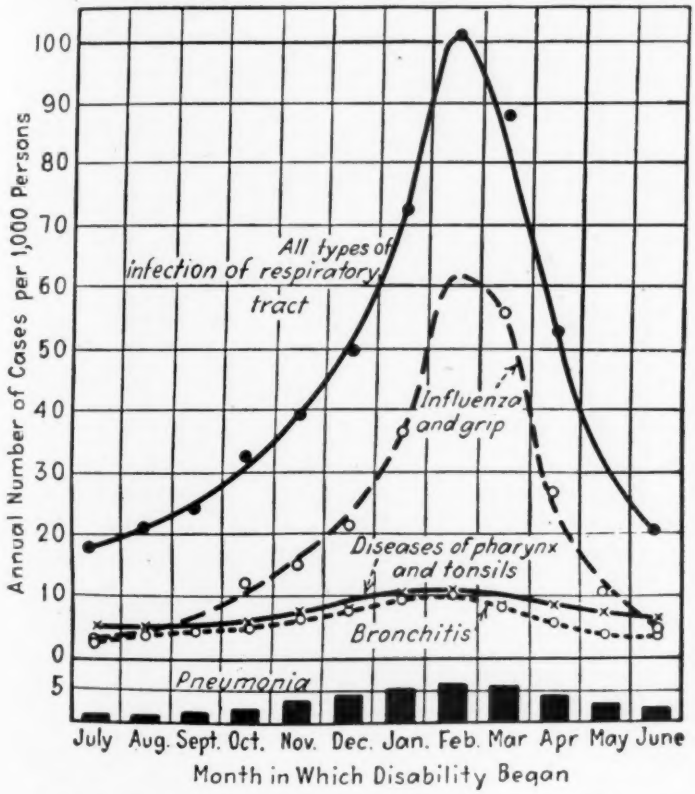

Fig. 2.—Incidence of infections of the respiratory tract by months.

cry is usually hoarse and a membrane is usually visible in the pharynx or on the tonsils. Supraglottic edematous laryngitis does not produce a croupy cough.

ETIOLOGIC ASPECTS

It is commonplace to search for one specific cause for a disease entity, but a little reflection will tell one that the summation of several factors is necessary to initiate a disease syndrome. This disease is no exception.

I believe that in winter the dry indoor air is one of the major predisposing factors, because it dehydrates mucous membranes, stops ciliary activity and thus permits organisms to penetrate the mucosa and set up inflammation. The curve in figure 2 showing the incidence of all respiratory infections follows the curve of household heating in winter. Table 1 showing the incidence of this disease by months indicates that most cases occur in winter.

The conus elasticus must be a particularly vulnerable portal of entry, because most of these patients had primary subglottic laryngitis. Table 2 demonstrates the fact that many had preceding head colds.

TABLE 1.—*Number of Cases in Each Month of a Five Year Period*

July	0	January	4
August	2	February	7
September	2	March	7
October	11	April	4
November	6	May	1
December	5	June	3

TABLE 2.—*Mode of Onset*

	Cases
Primary subglottic laryngitis	27
Previous head cold	21
Previous sore throat	1
Previous measles	1
Previous bronchitis	2

TABLE 3.—*Organisms Identified in Cultures Made from Endotracheal Exudate at the Time of Tracheotomy*

	Cases
Hemolytic Staphylococcus albus	7
Staph. albus and Streptococcus viridans	5
Nonhemolytic streptococcus	1
Hemolytic streptococcus and Staph. albus	1
No culture	1

Head colds cause nasal obstruction and mouth breathing, so that the extremely dry indoor air impinges directly on the larynx without having previously been moistened by passing through the nose. Undoubtedly, there is individual susceptibility to this disease, because 4 of these children had two attacks during successive winters and each time the disease began as primary subglottic laryngitis. A review of the case histories indicates that the bronchitis was secondary to the subglottic laryngitis. Richards [1a] and Farber in 1933 pointed out that the infection begins in the larynx and extends downward. Pneumonia did not develop in any patient in this series, owing, I think, to the fact that severe laryngeal obstruction was not permitted to continue.

The cultures studied by me and those of many other observers indicate that any of the pathogenic gram-positive cocci can be the bacterial agent responsible for this disease. Table 3 shows the organisms which were cultured from the exudate found in the trachea at the time a bronchoscope was introduced just prior to the tracheotomy. This exudate was aspirated by the bronchoscopic suction collector shown in figure 3; so there was no possible chance of contamination. Moreover, smears made from the exudate showed many gram-positive cocci mixed with the pus cells; so I think one goes out of one's way to postulate

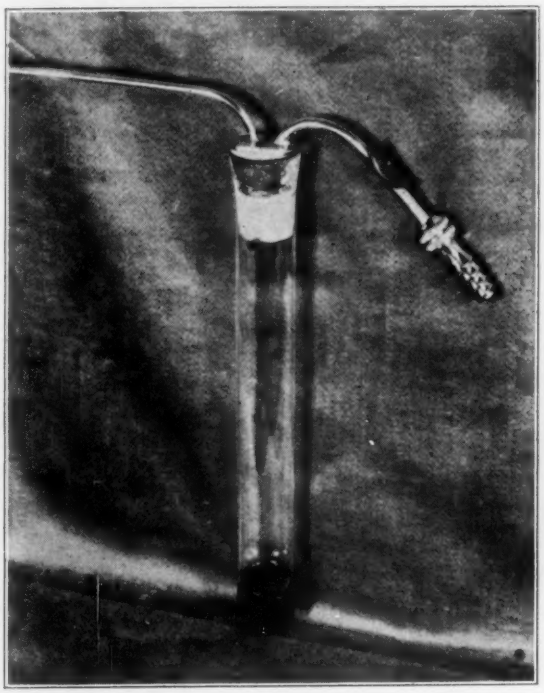

Fig. 3.—Bronchoscopic aspirating collector which furnishes an uncontaminated specimen of endobronchial exudate.

a virus infection as the cause of this disease. Font,[6] of Puerto Rico, reported 6 cases, in each of which a hemolytic staphylococcus was the infecting organism.

Males, for some unknown reason, are peculiarly predisposed to contract this disease. Of 52 patients, 39 were male, and 13 were female; that is, a 3 to 1 ratio. Of the patients requiring tracheotomy, 14 were male and 1 was a female. Perhaps there is more loose areolar connective tissue in the conus elasticus of males.

Table 5 showing the age incidence indicates that children between the ages of 1 and 2 years are most susceptible to this disease.

THE ENDOSCOPIC PICTURE

The pathologic changes observed by means of direct laryngoscopy and bronchoscopy were intense redness and swelling of the lateral walls of the trachea just below the vocal cords, so that the subglottic space was reduced to an anteroposterior slit about a millimeter wide. Often the subglottic tissues were coated with a film of dry adherent exudate, rough and gray, which extended downward a half inch below the level of the cords. The supraglottic structures were often inflamed but not edematous. Patients having supraglottic edematous laryngitis were not included in this series. The swelling of the subglottic tissues shown

TABLE 4.—*Sex Incidence in Fifty-Two Cases of Acute Laryngotracheobronchitis*

	Cases of Laryngo-tracheo-bronchitis	Cases in Which Tracheotomy Was Required
Male	39	14
Female	13	1

TABLE 5.—*Age Incidence*

	Cases of Laryngo-tracheo-bronchitis	Cases in Which Tracheotomy Was Required
Less than 1 year	9	1
1 to 2 years	19	7
2 to 3 years	12	4
3 to 4 years	7	3
4 to 5 years	3	
5 to 6 years	1	
6 to 7 years	1	

in figure 1 was firm and indurated, as was demonstrated by the fact that it always required considerable pressure to introduce even a 3.5 millimeter bronchoscope. This subglottic swelling was not due to simple watery edema but was due to inflammatory cells infiltrating the conus elasticus, as was shown by Richards [1] and Farber in 1933. Slow absorption of this infiltrate explains why it takes from one to two weeks for the croupy cough to clear up after the temperature has been normal.

As soon as a bronchoscope was introduced, a large amount of thick yellow pus was found in the trachea. The pus was there because it could not be coughed out through the slitlike lumen in the subglottic area. I agree with Galloway [5] that continuous postural drainage, both before and after tracheotomy, is important in the treatment of this disease.

TREATMENT

These patients were placed immediately in a humidified oxygen tent, and the last 24 were given penicillin, 15,000 to 25,000 units, by intramuscular injection, every two hours, day and night. Since I no longer use sulfadiazine in the treatment of this disease, it seems unnecessary to force fluids by gavage or by subcutaneous administration. The high humidity which can be produced in the oxygen tent by means of the mechanical humidifier [17] shown in figure 4 helps greatly to maintain the fluidity of the tracheobronchial secretions.

It should be remembered that penicillin is primarily a bacteriostatic and not a bactericidal agent. Therefore, some time may elapse before

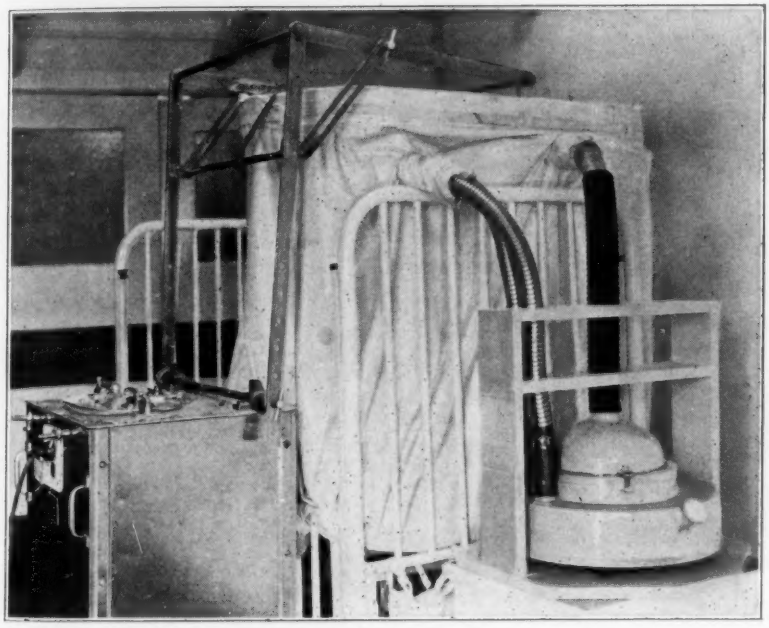

Fig. 4.—Mechanical humidifier adapted so that it may be used with an oxygen tent.

the invading organisms are killed by phagocytosis. It is interesting to note that many of these patients had relatively low total leukocyte counts, a fact which, in my opinion, helps to explain the severity of their infections. I think that a low leukocyte count is to be explained in these cases by a congenitally poor hemopoietic system, by poor nutrition or by impairment of the blood-forming organs due to effects of bacterial toxins. That penicillin may, however, act rapidly is shown by the

17. A similar humidifier adapted for use with an oxygen tent can be obtained from the Walton Laboratories, Irvington, N. J.

fact that, of the last 24 patients in this series who were treated with penicillin, 19 avoided tracheotomy. Of these 19, 14 showed a rapid drop in temperature and a decrease in the rates of the pulse and the respirations during the first twenty-four hours. One would not expect this rapid improvement if a virus was the infecting agent. The other 5 patients responded during the next twenty-four to forty-eight hours. The superiority of penicillin is indicated by tables 6 and 7. Of 5 penicillin-treated patients who required tracheotomy, 4 were sufficiently dyspneic to need tracheotomy as soon as they were admitted to the hospital, and the fifth required tracheotomy on the second hospital day. Delayed tracheotomy increases the probability of pneumonia due to lack of drainage of the tracheobronchial exudate. Early tracheotomy and postural drainage avoid the retention of exudate which causes pneumonia. Special day and night nurses are desirable before tracheotomy and are imperative after tracheotomy.

TABLE 6.—*Type of Drug Therapy*

	Cases of Laryngo-tracheo-bronchitis	Cases in Which Tracheotomy Was Required	
		Number	Per Cent
Sulfadiazine	28	10	36
Penicillin	24	5	21

TABLE 7.—*Average Number of Days in Hospital*

Sulfadiazine-treated group	10.0
Penicillin-treated group	7.8
Patients requiring tracheotomy	16.0

It has been stated that 50 per cent of tracheotomized patients die. I think this high mortality can be avoided if the methods of treatment subsequently outlined are followed. I agree with Baum [16] that some of the complications of this disease are man made. I think the crusting in the trachea and the bronchi which sometimes occurs after tracheotomy is due to the fact that the epithelium has been rubbed off by the bronchoscope and by the aspirating catheter. I insert a 3.5 millimeter bronchoscope just prior to tracheotomy, but since I have learned not to introduce the bronchoscope or the aspirating tube farther than 1 inch (2.5 cm.) beyond the cords, there has been no difficulty with crusting in the trachea or the bronchi. The cool, moist air provided by the humidified oxygen tent, described by me [18] in 1940, keeps the secretions sufficiently

18. Davison, F. W.: Treatment of Acute Laryngotracheobronchitis, Arch. Otolaryng. **32**:321 (Aug.) 1940; Some Observations on the Control of Temperature and Humidity in Oxygen Tents, Ann. Otol., Rhin. & Laryng. **49**:1083 (Dec.) 1940.

moist to prevent crusting in areas where there has been *no instrumental trauma.* At the time of bronchoscopy, just prior to tracheotomy, one never finds crusts in the trachea below the conus elasticus. The past two years my associates and I have not had to utilize bronchoscopic methods for removal of crusts after tracheotomy. During the preceding three years one patient required three bronchoscopic aspirations for removal of crusts, and one other patient required one similar procedure. The serous crusts which form as a result of abrasion of the mucosa contain fibrin and hence cannot be dissolved by any solvent which may safely be instilled through the tracheal cannula. However, isotonic solution of sodium chloride is used if the tracheal exudate is unusually viscid. The aspirating catheter used through the tracheal cannula is another frequent source of mucosal trauma. This trauma can and should be avoided by wrapping adhesive tape around the catheter as advocated by McCaskey,[11] so that the catheter cannot be introduced deeper than the end of the cannula.

TABLE 8.—*Duration of Laryngeal Obstruction Prior to Hospitalization in 15 Patients Requiring Tracheotomy*

	Patients	Number Requiring Immediate Tracheotomy
1 day	6	3
2 days	1	1
3 days	4	3
4 days	2	2
5 days	1	
7 days	1	1
Total		10

The two outstanding causes of death following tracheotomy are bronchial obstruction, which can be avoided by the measures just described, and pneumothorax, which can be avoided by proper performance of the tracheotomy. In one of my patients bilateral pneumothorax did develop. The cause of the dyspnea was promptly diagnosed by fluoroscopic examination. The air in the pleural cavities was aspirated, firm packing was placed around the tracheotomy tube and the pneumothorax did not recur. It is good practice, as advocated by Orton and associates,[8] to make a roentgen examination immediately after tracheotomy. Every one who contemplates doing a tracheotomy should read the articles by Neffson.[13]

The rapidity with which severe laryngeal obstruction develops in some cases is indicated by table 8 and implies that early treatment will not always prevent the need for tracheotomy.

If one does not fear the complications of tracheotomy, one will not temporize with measures which allow the exudate to be dammed back in the smaller bronchioles, with pneumonia and myocardial exhaustion resulting. Intubation was not used in any of these cases.

If the child's immunity is poor as evidenced by persistent fever, leukopenia or anemia, we give a transfusion, usually on the second or the third day, by the intramedullary route described by Tocantins, O'Neill and Price.[19] We have used the intramedullary route for the last three years and find it satisfactory. Ten transfusions were given in this series of 52 cases. Transfusion is our best means of rapidly increasing passive immunity.

SUMMARY AND CONCLUSIONS

Acute laryngotracheobronchitis usually begins as a primary subglottic laryngitis due to any of the pathogenic gram-positive cocci which cause other types of infection of the respiratory tract.

Most of these organisms are sensitive to the action of penicillin.

Because of the fulminating character of the infection, tracheotomy cannot always be avoided.

There were no deaths in this series of 52 consecutive cases, which included 15 patients who required tracheotomy.

The low mortality rate in this series is attributed to:

1. Cool, moist air provided by a humidified oxygen tent
2. Adequate doses of penicillin or sulfadiazine, preferably the former
3. Avoidance of instrumental trauma to the mucosa of the trachea and bronchi
4. Postural drainage
5. Early tracheotomy.
6. Transfusion when indicated
7. Careful personal attention to each patient

I wish to acknowledge the skilful assistance of Dr. John G. Wilcox in the care of these patients.

19. Tocantins, L. M.; O'Neill, J. F., and Price, A. H.: Infusions of Blood and Other Fluids into General Circulation Via Bone Marrow in Traumatic Shock and Other Forms of Peripheral Circulatory Failure, Ann. Surg. **114:** 1085 (Dec.) 1941.

DISTURBANCES OF OLFACTION RESULTING FROM INTRANASAL USE OF TYROTHRICIN

A Clinical Report of Seven Cases

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AND
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SINCE 1941 the literature abounds in glowing clinical reports of the beneficial effects of the topical use of tyrothricin, especially in the field of otolaryngology. Crowe and associates [1] since 1942 have made many contributions to the literature describing this as a safe bactericidal substance especially efficient in infection of the sinuses. This observation has been confirmed and reported by others over the past six years. This has led many of the leading pharmaceutic houses to market preparations containing various concentrations of this material for use as nose drops. With the earlier preparations difficulty was experienced in keeping the solution stable and potent. This has been overcome in the past year or so by the addition of various preservatives, and physicians have been heavily detailed on the excellent qualities of these solutions. We ourselves were convinced of the efficacy of these solutions and until recent months had been enthusiastically prescribing two such preparations for nasal and sinus infection.

However, in the past six months we have observed a number of cases of distressing disturbances of the olfactory sense, which we are convinced are attributable to the intranasal instillation of these two preparations. We are here reporting 7 such cases. We are sure that we have observed additional cases, but they had been lost in our files before we were fully aware of the nature of the condition and we have therefore not been able to document them fully. We had not intended to report these cases for another six months or so, since none of the

1. Bordley, J. E.; Crowe, S. J.; Dolowitz, D. A., and Pickrell, K. L.: The Local Use of Sulfonamides, Gramicidin (Tyrothricin) and Penicillin in Otolaryngology, Ann. Otol., Rhin. & Laryng. **51**:936 (Dec.) 1942. Crowe, S. J.; Fisher, A. M.; Ward, A. T., Jr., and Foley, M. K.: Penicillin and Tyrothricin in Otolaryngology, ibid. **52**:541 (Sept.) 1943. Crowe, S. J., and Ward, A. T., Jr.: Local Use of Sulfadiazine, Tyrothricin, Penicillin and Radon in Otolaryngology, North Carolina M. J. **4**:431 (Oct.) 1943. Crowe, S. J.: The Local Use of Sulfadiazine Solution, Radon, Tyrothricin and Penicillin in Otolaryngology, Ann. Otol., Rhin. & Laryng. **53**:227 (June) 1944.

patients have as yet made a complete clinical recovery, but they are showing signs of gradual improvement. The recent report of Otenasek and Fairman [2] of 2 tragic cases in which chemical meningitis resulted when this substance was used in irrigation of the frontal sinuses, and their statement that they produced similar reactions in experimental animals, has stimulated us to make a preliminary clinical report of these cases of olfactory disturbances. These effects are fortunately not as severe or threatening as those of the Baltimore observers, but are, none the less, most disagreeable to the patient and equally embarrassing to the physician. For this reason we feel it mandatory now to call these effects to the attention of members of the profession at large, and perhaps encourage others to look for and report similar results.

The pharmacologic and toxicologic aspects of tyrothricin have been thoroughly studied and reported by Herrell and Heilman, Rammelkamp and Weinstein, and Robinson and associates and others since the original work of Dubos [4] in 1939. Tyrothricin was found to be a definite protoplasmic poison, its effects manifested principally in the form of hemolysis, if administered by any parenteral method. It was found, however, to be apparently safe in active bactericidal concentration for topical application or for instillation into body cavities if the latter action was not too prolonged. We can reason only by conjecture on the effect herein reported, but it would appear to be a deleterious reaction of the neural elements of the olfactory mucous membrane.

Two different preparations were used clinically and will be referred to here as preparations 1 and 2. Both contained tyrothricin in concentrations of approximately 1 : 5,000. We report 4 cases resulting from the use of preparation 1 and 3 from the use of preparation 2. The solutions were applied both as spray and as drops.

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2. Otenasek, F. J., and Fairman, D.: Chemical Meningitis Following Use of Tyrothricin, Arch. Otolaryng. **47**:21-28, (Jan.) 1948.

3. (*a*) Herrell, W. E., and Heilman, D.: Experimental and Clinical Studies on Gramicidin, J. Clin. Investigation **20**:583 (Sept.) 1941. (*b*) Rammelkamp, C. H., and Weinstein, L.: Hemolytic Effect of Tyrothricin, Proc. Soc. Exper. Biol. & Med. **48**:211 (Oct.) 1941; (*c*) Toxic Effects of Tyrothricin, Gramicidin and Tyrocidine, J. Infect. Dis. **71**:166 (Sept.) 1942. (*d*) Robinson, H. J., and Molitor, H.: Some Toxicological and Pharmacological Properties of Gramicidin, Tyrothricin and Tyrocidine, J. Pharmacol. & Exper. Therap. **74**:75 (Jan.) 1942. (*e*) Robinson, H. J., and Graessle, O. E.: In Vitro and in Vivo Studies of Gramicidin, Tyrothricin and Tyrocidine, ibid. **76**:316 (Dec.) 1942.

4. Dubos, R. J.: Studies on a Bactericidal Agent Extracted from a Soil Bacillus, J. Exper. Med. **70**:1 (July) 1939; The Effect of Specific Agents Extracted from Soil Micro-Organisms upon Experimental Bacterial Infections, Ann. Int. Med. **13**:2025 (May) 1940.

While this report was being prepared, a discussion with one of our local colleagues, Dr. Earl D. Carter, revealed that a similar condition had been observed in 1 of his patients, and this case is being included in our series (case 7). He stated that he had prescribed tyrothricin preparations only three times, and in 1 of these 3 cases anosmia developed.

REPORT OF CASES

CASE 1.—J. H., a man aged 47, was seen in June 1947, just before he left to spend his summer vacation in Colorado. He was suffering from acute nasopharyngitis and mild acute sinusitis. Preparation 1 was prescribed, to be used with a jetomizer. He was not seen again until the end of the summer, when he stopped in the office to seek help for his perverted sense of smell and taste. He reported that three to four days after he started to apply the spray he noted a complete loss of his sense of smell and taste. He attributed this to the respiratory infection and continued to use the nasal medication for several more days. By this time the nasal symptoms had subsided, and the medication was discontinued. However, the anosmia continued but at the end of about a month was gradually improving when another "cold" developed, and he began using the medicament again. Within a day complete anosmia returned, and, suspecting the medicine, he discontinued its use. He was completely devoid of olfactory and gustatory sense for about a month, when he began to notice unpleasant and obnoxious odors. All food, particularly highly spiced and aromatic dishes, had a repugnant odor and made eating an unpleasant and distasteful experience. These symptoms persisted. He was seen on his return in the fall and was followed for several weeks, without change in these symptoms. He was then transferred to another community, and further observation has been impossible.

CASE 2.—E. P., a woman aged 32, presented herself, Oct. 10, 1947, with a history of a "cold" of two weeks' duration, some frontal headaches and profuse, foul, lemon yellow nasal discharge. Examination showed free pus in both nostrils and general clouding of the sinuses to transillumination. Acute rhinitis and sinusitis was diagnosed. She was given local treatment in the office, and preparation 2 was prescribed, to be instilled intranasally at home. She was not seen again until Nov. 1, 1947, when she returned to state that the headaches had disappeared and that the nasal congestion and discharge had about stopped. However, she stated that on about the third day after starting the intranasal medication she noted complete loss of the sensation of smell and taste. Thinking this was due to the infection, she had continued to use the drops as directed. Intranasal medication was stopped at this visit. The infection gradually subsided. Complete anosmia continued until the latter part of January, when she noted a gradual onset of perverted odors. These became increasingly annoying for about three weeks, to the point that she had to take to her bed; she was unable to eat because of the offensive odors of all foods, which induced severe nausea and occasional vomiting. This state of affairs became so alarming that her family physician seriously considered taking her to the hospital to combat her malnutrition. Within the past two weeks she has gradually improved and is now able to take most bland, nonaromatic foods. She remarked that tobacco smoke caused the most offensive of all odors. She still experiences extremely obnoxious sensations of smell from tea, coffee, cocoa, cokes and all soft drinks, and all spices.

CASE 3.—J. H., a woman aged 34, a personal friend of one of us, called by telephone for advice as to treatment of an annoying acute coryza. Preparation 2

was suggested and was used as drops. Immediately after the first instillation she noted marked perversion of her sense of smell, particularly for cigaret smoke. She attributed this to the "cold" and continued to use the drops for another three days. By this time her sense of smell was so badly perverted that she could barely force herself to eat, and she stopped smoking because the odor was so disagreeable. The nasal infection cleared in a few weeks, but the parosmia has continued for the past five months, to the present. Within the past month her symptoms have improved somewhat—enough that she has again started smoking but still finds the odor of hot, highly spiced food, "coca colas," chocolate and onions very distasteful.

CASE 4.—G. K., a man aged 74, had been under observation for two months because of an idiopathic paresis of the left vocal cord of about six months' duration. He presented himself on Oct. 27, 1947 for routine examination of his larynx, complaining of an acute sore throat and nasal congestion. He was found to be suffering from acute nasopharyngitis, and his nasal smear was loaded with pus. The vocal cord was found to be regaining some motion at this time. Preparation 1 was prescribed for intranasal use. He was not seen again until December 17, when he reported that about two weeks after starting to use the nose drops he noticed a loss of sense of taste and of smell for some odors and an obnoxious perversion of smell for the others. He has been seen at intervals since, and the parosmia has persisted to the time of writing—a period of almost four months. Within the past few weeks, a few odors have been perceived as normal, but most odors still impress him as having a perverted and offensive nature, although the intensity of such perversions is not as great. During the interval, all evidence of nasal and sinus infection has cleared, and the original complaint of the voice has improved considerably. The cords now move almost normally.

CASE 5.—R. G. K., a woman aged 30, presented herself on Oct. 24, 1947, with a history of "bronchitis since childhood" and tightness and some wheezing in the chest for the past three years. Occasionally she had some purulent secretion from the nose, and a history of frequent nasal congestion and sneezing throughout most of the year. She had been advised to have her nose and throat examined for a possible focus of infection as the cause of the symptoms related to the chest. The septum was slightly deflected to the left, and there was some free mucopus in the middle meatus. Transillumination showed the sinuses essentially normal. Mucopus was seen on the posterior pharyngeal wall. A nasal smear showed a large quantity of fresh pus but no eosinophils. Because of the history, a diagnosis of asthma, nasal allergy and nasal infection was made. The patient was scheduled for skin tests and given preparation 1 for intranasal use, as drops. She returned three weeks later for cutaneous tests and reported that a week after starting the nasal medication she noticed loss of her sense of taste and smell. She continued, however, to use the drops until told to stop at this time. She had never had any previous disturbance of smell. At this visit she was tested with an intravenous injection of camphor water U.S.P. There was no olfactory response to this, a result proving that the anosmia was of an essential and not a respiratory type. Shortly after this she began to smell disagreeable and offensive odors. This has continued to the present time, with some lessening of the intensity of the parosmia. Recently she complains more bitterly of the "off" taste of most foods than she does of the abnormal odors. Her nasal allergic symptoms have cleared up almost entirely during this time, on a regimen of restrictive diet and elimination of offending inhalants. The nasal infection has also cleared up satisfactorily.

CASE 6.—J. F., a woman aged 33, was seen first on Oct. 28, 1947, because of nasal congestion and frontal headache of about two weeks' duration. She had been subject to such conditions on infrequent occasions for the past six years. The past history was essentially irrelative except for some sneezing during the preceding summer. Her nasal smear at this time showed some fresh pus but no eosinophils. Preparation 2 was prescribed as nose drops. She was seen about ten days later, at which time she reported improvement of her nasal symptoms, but stated that there had been complete loss of the sense of smell for the preceding four days. There was still some pus in the nose. The offending preparation was stopped, and another preparation, containing ephedrine and sulfadiazine, was prescribed. About a week later she was again seen, and her nose had cleared well. She was beginning to perceive some odors, but these seemed of an abnormal nature. All medication was then stopped, and she has remained about the same to the time of writing, a period of about four months. The intensity of her parosmia has lessened to some degree, but there are still some foods that she cannot smell at all, and those that she can smell impress her as abnormal and disagreeable.

CASE 7.—V. L., a woman whose age is not known, was seen on Jan. 20, 1948, with signs and symptoms of an acute cold. A slight diminution of the sense of smell accompanied this cold. Preparation 2 was prescribed as nose drops. After five instillations, she noted complete loss of her sense of smell. The cold promptly cleared up but the anosmia has continued to the time of writing (two months).

ADDENDUM

A patient was in the office on the day that this report was being prepared to mail for publication. Her history is being added to this series because her symptoms are of longer duration than those noted in any of the cases presented in the foregoing section of this paper.

J. E. M., a woman aged 48, was seen in consultation July 8, 1947, complaining of deafness and vertigo. An incidental finding in the examination at that time was an acute nasal and sinus infection. Preparation 1 was prescribed for relief of this condition. She was not seen until March 20, 1948, when she returned for a check on her hearing and gave the following story concerning her sense of smell. She used the prescription in her nose as drops for two or three weeks and noticed during this time that she had completely lost her sense of smell and taste. Her nasal symptoms had cleared up by this time, but the complete anosmia continued until October, when she began to note strange and exceedingly unpleasant odors. She noticed this particularly when around fried or greasy foods. She was frequently nauseated by the odors but never vomited. Eating became difficult, and she has lost 20 pounds (9 Kg.) in weight. She states that she had been fond of eggs and usually ate a large number of them but that since the onset of the anosmia they smell like "creosote," and she has not been able to force down a single egg. She states that she cannot describe the other odors—"they smell like nothing that I have ever smelled before." In the past two months she has noticed that tart foods, especially jellies, have about normal smell and taste, and she has existed almost wholly on foods of this type. Tobacco smoke is especially unpleasant, and she has had to ask her husband not to smoke in the house. She has had no other colds or influenza during this period. Her symptoms have now persisted for over eight months, and she states that the intensity of the parosmia is still severe.

COMMENT

We have presented here 8 cases in which anosmia or parosmia developed immediately, or shortly after, a solution of tyrothricin began to be used in the nose. These symptoms have been persistent, ranging in duration from four to eight months. The symptoms have outlasted by months the condition for which the solutions were originally prescribed. None of the patients were suffering from clinical influenza at the time of use of the solutions. One patient had a definite allergic history and was shown by smear and skin tests to have a perennial allergy. However, she had never had any disturbance of olfaction prior to the use of the solutions, and the parosmia lasted long after the nasal allergic symptoms were brought under control.

One patient (case 5) was tested by injecting camphor water U.S.P. intravenously and showed no response. This would definitely place her condition in the category of an essential anosmia.

None of the patients have regained normal sense of smell. Concomitant disturbance of taste is also complained of by the patients. Most of the patients think there has been some improvement, at least as far as intensity of the parosmia is concerned. We are continuing to observe these patients and will report further on the final outcome of their disease.

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BLAST INJURIES OF THE EARS

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THE CONCUSSION or blast produced by the high explosives used in modern warfare is greatly injurious to the auditory apparatus. This fact quickly became obvious to an otolaryngologist treating battle casualties in World War II. On my service in one of the United States Army general hospitals in England more patients were seen with blast injury of the ears than with any other injury or disease. The studies reported here were made on 100 patients with acoustic trauma admitted to this general hospital in the first six months after the invasion of the European Continent in June 1944. These were the first 100 patients who could be adequately studied, although many others were seen and treated during this period. The patients not included in this series were eliminated because of other injuries which prevented audiometric examination or because they were under observation for too short a time. Many of these patients were seen within one week of injury (table 1), but none before the third day. Some of them remained under observation for as long as three months (table 2). The findings in these men were deafness, tinnitus, interstitial hemorrhage and rupture of the tympanic membrane, and acute suppurative otitis media.

DEAFNESS

Whispered voice tests and audiometric studies were done on all these patients and were repeated at intervals to follow their progress. A rough determinaton of the acuity of hearing for speech can be obtained by the whispered voice test. Audiometry is a much more accurate method of evaluating and following changes in the loss of hearing for sound. This is borne out by comparing the findings made by audiometry with those obtained by whispered voice and tuning fork tests (table 3).

A history of aural disease which existed prior to the blast injury was given by 22 of these patients. Six had total unilateral deafness,

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11 had partial deafness and 9 had healed acute suppurative otitis media. Four gave a history of both deafness and previous aural suppuration.

Seventy per cent of the injuries were caused by high explosive shells. In some cases there had been repeated exposure to blast and heavy firing with no acute injury occurring (table 4).

The impairment of hearing was severe in 14 patients, moderate in 15 and slight in 71.

Mechanism.—Zuckerman [1] described the blast wave as a single pulse of increased pressure followed by a phase of suction. The waves move extremely rapidly, much more rapidly than sound close to the explosion. The total duration of the whole wave at any given point within 200

TABLE 1.—*Time Elapsing Between Injury and Observation*

	Patients
Less than 1 week	26
Between 1 and 2 weeks	20
Between 2 weeks and 1 month	31
Between 1 and 2 months	16
Between 2 and 3 months	5
Between 3 and 4 months	1
Between 4 and 5 months	1
Total	100

TABLE 2.—*Length of Observation*

	Patients
1 week	32
1 month	38
2 months	23
3 months	7
Total	100

feet of an explosion is brief. Thus, at 30 feet from a 70 pound charge the pressure component lasts about five and the suction component thirty milliseconds. The duration of the pressure component increases progressively with increasing distance from the charge, but the suction component remains more or less constant.

According to Collins,[2] the intrinsic muscles of the middle ear cannot exert any considerable function in protecting the middle or the inner ear against blast because the full extent of the "damping action" is not developed until some thirty milliseconds after the onset of the stimulus.

1. Zuckerman, S., in Discussion on Problem of Blast Injuries, Proc. Roy. Soc. Med. **34**:171-192 (Jan.) 1941.

2. Collins, E. G.: Injuries to the Ears Among Battle Casualties of the Western Desert, J. Laryng. & Otol. **59**:1-15 (Jan.) 1944.

The fact that the cochlea is not thus protected against blast concussion waves explains its vulnerability as demonstrated in the following case histories.

CASE HISTORIES

The audiograms of patients A to G, inclusive, are presented because each demonstrates the characteristic curve obtained when the blast did not rupture the tympanic membrane.

Patient A, a corporal in the infantry, aged 27, was first seen, Sept. 14, 1944, complaining of tinnitus (frying noise) in both ears. This had been present since July 26, when he was wounded by the explosion of an 88 mm. shell. He had been

TABLE 3.—*Comparison of Findings with Whispered Voice and Tuning Fork Tests and with Audiometry*

Deafness	Whispered Voice and Tuning Fork Tests	Audiometry
Unilateral	30	8
Bilateral	62	92
None	8	0
Total	100	100

TABLE 4.—*Explosives Causing the Injuries*

	Patients
High explosive shell (type undetermined)	35
88 mm. shell	18
Mortar shell	17
Land mine	5
Rifle grenade	1
Shell and trinitrotoluene (TNT)	1
Concussion grenade	1
Aerial torpedo	1
Hand grenade	1
No acute injury	14
Cause undetermined	6
Total	100

exposed to heavy firing for one month before injury. There was no complaint of deafness. Nothing in the past history appeared contributory. Examination revealed normal tympanic membranes. Hearing for whispered voice was: right 15/15, left 15/15. The Rinne test showed that air conduction was better than bone conduction bilaterally. The audiogram was made on September 14 (fifty days after injury).

Patient B, a private in a tank battalion, aged 20, was first seen, Aug. 15, 1944, because of ringing and discomfort in the right ear and ringing in the left ear since being exposed to shell blast on July 18. There was no complaint of deafness. The past history was irrelevant. Examination revealed a slightly thickened right tympanic membrane; the left was normal. Hearing for whispered voice was: right 15/15, left 15/15. The Rinne test showed that air conduction was better than bone conduction bilaterally. The audiogram was made on August 31 (forty-two days after injury).

Patient C, a corporal in an armored regiment, aged 25, was first seen, Aug. 16, 1944, because of ringing in the ears. There was no complaint of deafness. He saw action in Sicily and was in action for fifty-seven days in France. The rest of the past history was not related to the tinnitus. Examination revealed normal tympanic membranes. Hearing for whispered voice was: right 15/15, left 15/15. He was evacuated from the front on July 28, and the audiogram was made on August 31.

Patient D, a sergeant in the armored infantry, aged 28, was first seen on Nov. 5, 1944; he complained that there had been ringing in both ears, more severe on the left, since a mortar shell landed about 3 yards (about 3 meters) from him on October 9. Nothing in his past history appeared contributory. Examination revealed normal tympanic membranes. Hearing for whispered voice was: right 15/15, left 15/15. The Rinne test showed that air conduction was better than bone conduction bilaterally. In the Weber test the sound of the vibrating fork held against the forehead in the midline was referred to the right. The audiogram was made on November 5 (twenty-six days after injury).

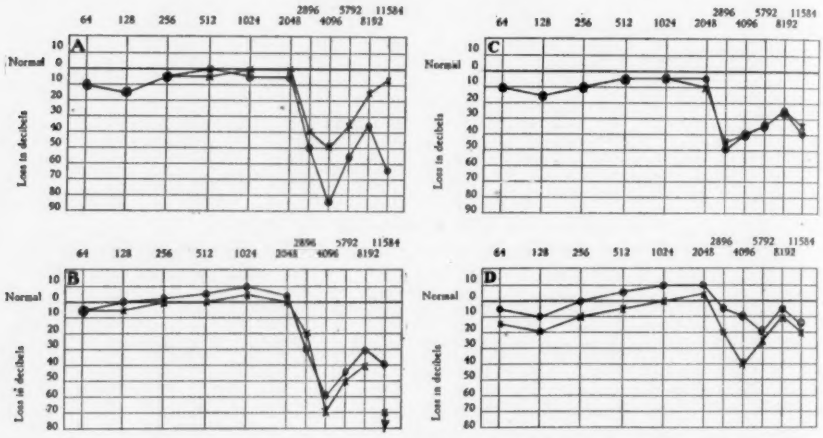

Chart 1.—Audiograms of patients A, B, C and D. These show the characteristic curve obtained when the blast did not rupture the tympanic membrane. Crosses represent the threshold of hearing of the left ear; circles, that of the right ear. In all 4 cases the whispered voice test showed hearing 15/15 in both ears.

The audiograms of patients A, B, C and D (chart 1) show the abrupt dip or gap at or near frequency 4096 which Bunch [3] has described as characteristic of the deafness of ears that have been excessively stimulated by industrial noises or repeated excessive explosive sounds. The hearing of whispered voice was 15/15 in both ears in all 4 cases. Another finding was a slight loss in the low tones (below frequency 512).

3. Bunch, C. C.: Symposium: The Neural Mechanism of Hearing; "Nerve Deafness" of Known Pathology or Etiology; the Diagnosis of Occupational or Traumatic Deafness; a Historical and Audiometric Study, Laryngoscope **47**:615-691 (Sept.) 1937.

Patient E, a private in the infantry, aged 31, first seen on Sept. 9, 1944, complained of ringing in both ears. This had been present since June 13 when, in France, a mortar shell landed just behind him. His past history offered nothing relative to the condition. Examination revealed normal tympanic membranes. The hearing of whispered voice was: right 15/15, left 12/15. The Rinne test showed that air conduction was better than bone conduction bilaterally. In the Weber test sound was not referred to either ear. The audiogram was made on September 9 (ninety-eight days after injury).

Patient F, a sergeant in the armored infantry, aged 25, was first seen on Oct. 20, 1944, when he complained that there had been deafness and ringing in the right ear since June 29 when, in France, a shell landed near his right side. After a period of hospitalization, he returned to duty until September 30; on that date he received multiple shell fragment wounds of the right arm and leg as a result of a mortar shell explosion. The past history was irrelevant. Examination revealed normal tympanic membranes. The hearing of whispered voice was: right ½/15,

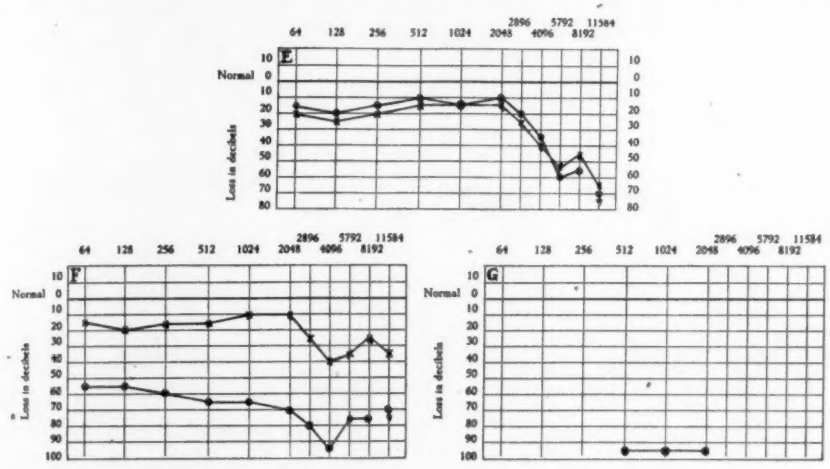

Chart 2.—Audiograms of patients E, F, and G. The tympanic membranes of these patients were not ruptured. Crosses represent the threshold of hearing of the left ear; circles, that of the right ear.

left 15/15. The Rinne test showed that air conduction was better than bone conduction bilaterally. In the Weber test sound was referred to the left. The audiogram was made on November 21.

Patient G, a private in the infantry, aged 24, was first seen, June 13, 1944, because of almost total deafness. He had been injured in action on June 7, in France, when a shell landed about 15 feet (about 5 meters) from him. He lost consciousness for one and a half hours and on recovery noted that he was unable to hear. There was no bleeding from the ears or tinnitus. The past history was as follows: In May 1942 while he was in the mess hall of a military post in the United States, the building was struck by lightning. As a result of this accident, his hearing was considerably impaired. His hearing improved during six weeks of hospitalization in the station hospital at the post, but it never returned to its previous level.

Examination revealed normal tympanic membranes. The hearing of whispered voice was 0/15 in both ears. The 512 tuning fork being used, air conduction was

found to be markedly diminished bilaterally, and no bone conduction was present. No improvement occurred during his hospitalization of thirty-nine days. The audiogram was made on July 2 (twenty-five days after injury).

Patient E's left ear shows a loss of from 15 to 25 decibels in the frequencies below 4096. The high tones show a more severe loss. When tested with whispered voice the hearing of this ear was 12/15.

The audiometric curve recorded for the right ear of patient F is consistent with a severe impairment of hearing. The whispered voice indicated that hearing was ½/15. The high tones show the greatest loss, and there is an additional dip at frequency 4096.

Patient G demonstrates the severe or extreme type of traumatic deafness which can occur with no gross injury being done to the tympanic membrane or the middle ear.

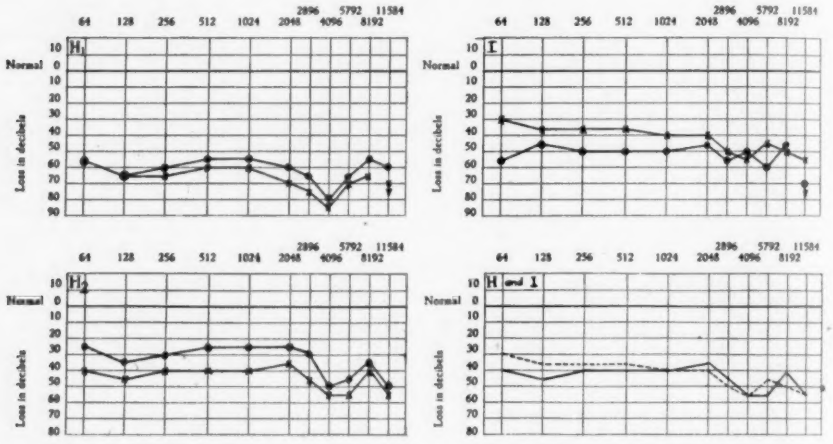

Chart 3.—Audiograms of patients H and I. They demonstrate the findings associated with traumatic rupture of the tympanic membrane. The two audiograms of patient H were taken eight and forty-two days after his injury, respectively. At eight days the whispered voice test showed hearing 1/15 in both ears. At forty-two days it showed hearing 12/15 in the left and 13/15 in the right ear. *H* is indicated by the solid line; *I*, by the dotted line.

The hearing of patient I when tested with whispered voice two months after his injury was 0/15 in both ears.

A comparison of the audiometric curves of the left ears of the two patients shows their similarity. Yet there was a significant difference in the ability to understand speech, as determined by whispered voice tests: The hearing of patient H was 12/15; that of patient I, 0/15.

The audiograms of all the patients presented (A to G inclusive) show that the critical frequencies for hearing speech (512, 1024 and 2048) were the frequencies heard best.

In the following cases the audiograms demonstrate the findings associated with traumatic rupture of the tympanic membrane.

Patient H, a private in the infantry, aged 34, first seen on Aug. 6, 1944, complained of ringing in his ears and deafness, present since he was injured in action by a mortar shell explosion on August 1, in France. At that time he lost consciousness for about five minutes. On recovering consciousness he was deaf, there was ringing in his ears and he was bleeding from the nose, the face and the ears. Since the time of injury there had been some improvement in hearing. Nothing in the past history was contributory. Examination revealed small anterior-inferior perforations of both tympanic membranes. The hearing tested with whispered voice was: right 1/15, left 1/15. The Rinne test showed that air conduction was better than bone conduction bilaterally, and in the Weber test sound was referred to the left. An audiogram made on August 9 (eight days after injury) showed a marked loss of hearing. Another audiogram was made on September 12, after the tympanic membranes had healed (forty-two days after injury). At that time, as tested with whispered voice, hearing was: right 13/15, left 12/15.

Patient I, a private in the infantry, aged 35, first seen on Nov. 23, 1944, complained that there had been deafness and ringing in his ears since September 22, when three 88 mm. shells exploded near his foxhole. A central perforation of the left tympanic membrane had been recorded by a previous examiner. The past history was irrelevant. Examination revealed a thickened left tympanic membrane with a healed small central perforation. The right tympanic membrane was

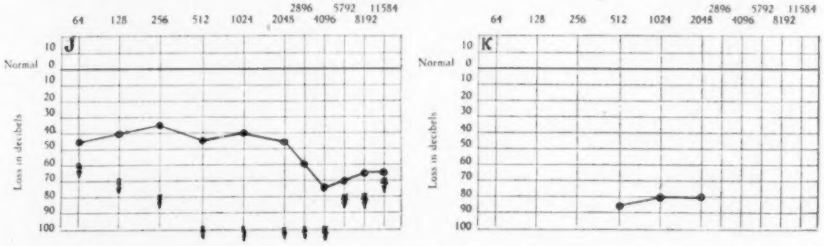

Chart 4.—Audiograms of patients J and K, who had a severe type of traumatic deafness, with loss of all useful hearing. In each of these patients the left tympanic membrane was ruptured; the right was intact. The audiogram of patient J was made twenty-eight days after injury, when the whispered voice test showed hearing to be ½/15 in the right ear and 0/15 in the left. The audiogram of patient K was made thirty days after injury, when the whispered voice test showed hearing 0/15 in both ears (circles represent the right, crosses the left, ear).

normal. The Rinne test showed that air conduction was better than bone conduction bilaterally. In the Weber test sound was not referred. The whispered voice test showed that hearing was: right 0/15, left 0/15. The audiogram was made on November 23 (fifty-two days after injury).

Patient H demonstrates an early severe loss of hearing that was present five days after a blast injury which caused rupture of both tympanic membranes. There was a moderate final loss of hearing forty-two days after the injury, after the tympanic membranes had healed.

The audiograms made for the left ears of patients H and I are quite similar; yet there is a significant difference in the ability to understand speech in these two patients, as determined by the whispered voice test. With that test, the hearing of the former was 12/15 and the hearing of the latter 0/15. This demonstrates the difficulty that

arises when one attempts to anticipate from the audiogram a patient's ability to understand speech.

Patient J, a private in the infantry, aged 30, first seen on Oct. 25, 1944, complained that there had been deafness and ringing in his ears since September 27, when an 88 mm. shell exploded about 6 feet (about 2 meters) from his foxhole. He was completely deaf for the first week following the injury, after which slight improvement of hearing was noted in the right ear. The past history revealed no contributory factor. The right tympanic membrane was found normal. There was a large anterior-inferior marginal perforation of the left tympanic membrane; no suppuration was present. With whispered voice, the hearing was: right ½/15, left 0/15. The audiogram was made on October 25 (twenty-eight days after injury).

Patient K, a sergeant in the infantry, aged 24, was first seen on Oct. 30, 1944. He complained of deafness, ringing in the ears, headache and earache, present since he was injured in action on October 9, in France, when a land mine exploded 20 feet (6 meters) from him. Since the injury he had been able to blow air out of the left ear. The past history was irrelevant. The right tympanic membrane was normal. The left tympanic membrane showed two small clotted areas which were considered healing perforations. With whispered voice, hearing was: right 0/15, left 0/15. The 512 tuning fork was heard only by air conduction on the right and was not heard on the left. The audiogram was made on November 8 (thirty days after injury).

Patients J and K demonstrate again a severe type of traumatic deafness, with loss of all useful hearing. In each of these patients the left tympanic membrane was ruptured; the right was intact.

Patient L, a captain, was first seen on July 28, 1944, with complaints of deafness and ringing in both ears. He was injured in action July 25, in France, when a shell and TNT exploded about 25 feet (7.5 meters) from him. He noted deafness and ringing in both ears immediately. He had had tinnitus, however, for four days prior to the injury as a result of taking quinine for malaria. The past history was otherwise unrelated to the auditory impairment. The right tympanic membrane had a small anterior-superior perforation of the pars tensa and an interstitial hemorrhage in its lower half. The left tympanic membrane showed what appeared to be two perforations, anterior and posterior, with the remainder of the membrane covered by blood clot. Tested with whispered voice the hearing was: right 3/15, left 2/15. Hearing improved rapidly in the right ear, and on August 2 it was shown by whispered voice test to be: right 13/15, left 2/15, at which time the first audiogram was made (eight days after injury). When the clot seen in the region of the left tympanic membrane absorbed, there was a large central perforation with destruction of the lower three fourths of the pars tensa and loss of part of the handle of the malleus. The perforation of the right tympanic membrane healed rapidly, and the drum cleared. On the left the perforation showed no tendency to heal and the hearing remained 2/15. The audiogram made on September 18 (fifty-five days after injury) showed considerable improvement in the hearing of the right ear but little change in that of the left. By whispered voice test the hearing was: right 15/15, left 2/15.

The following case history demonstrates the findings in a patient who had rupture of both tympanic membranes and bilateral acute suppurative otitis media.

Patient M, a private in the infantry, aged 33, first seen on June 20, 1944, complained that there had been deafness, ringing in and drainage from both ears since he was injured in action June 10, in France, by a shell explosion. Following the explosion, he was unable to hear. On June 13 a notation was made on the patient's record that both ear drums were ruptured. The past history was irrelevant. The tympanic membranes were reddened and thickened, with a small anterior-inferior perforation present in each of them. A small amount of purulent drainage was present bilaterally. With whispered voice the hearing was: left 1/15, right 0/15. The Rinne test, made with the 512 tuning fork, showed that air conduction was better than bone conduction bilaterally. In the Weber test sound was not referred to either ear. Local aural therapy was administered, and a course of sulfadiazine was given from June 21 to 29. The right ear became dry on June 24 and the left on June 27. The perforations healed, and then the ears were treated by inflation and pneumomassage. On July 20 the hearing was: left 15/15,

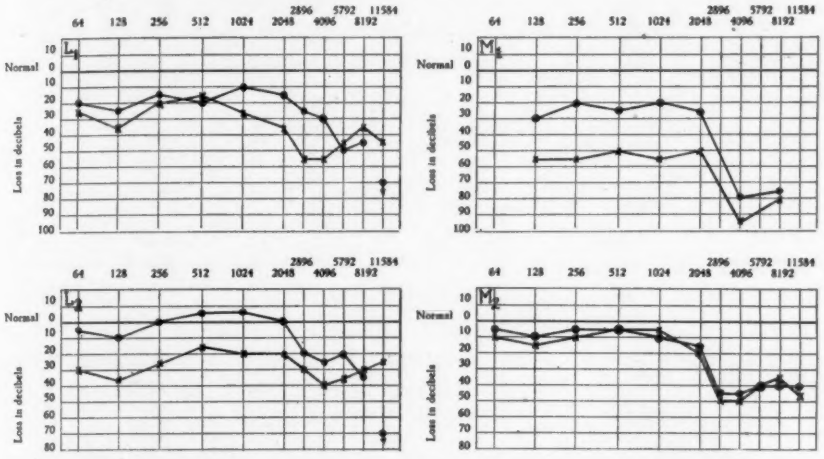

Chart 5.—Audiograms of patients L and M. The first audiometric study of L (made eight days after his injury) demonstrates the mixed type of deafness occurring after the tympanic membranes have been ruptured. The first study of M (made fourteen days after his injury) demonstrates the same type of deafness occurring with rupture of the tympanic membranes and acute suppurative otitis media. The whispered voice test showed L's hearing to be: right 3/15, left 2/15, and M's hearing, right 1/15, left 0/15. Patient L's right ear (circles) and both ears of patient M show almost complete recovery except for the higher frequencies (above 2048) after healing had occurred (fifty-five and forty days after injury, respectively).

right 15/15. The first audiogram, made on June 24 (fourteen days after injury), showed a mixed type of curve. The final audiogram was made on July 20 (forty days after injury), after healing of the drums and when it was believed that the maximum recovery had occurred.

The first audiometric study done on patient L demonstrated the mixed type of deafness occurring after traumatic rupture of the tympanic membranes. The first audiometric study done on patient M demonstrated the same type of deafness occurring with rupture of the tympanic membranes and acute suppurative otitis media. Patient L's

right ear and both ears of patient M show almost complete recovery except in the higher frequencies (above 2048) after healing had taken place.

RUPTURE OF THE TYMPANIC MEMBRANE

Rupture of the tympanic membrane occurred in 31 patients (31 per cent). It was unilateral in 20 and bilateral in 11. Considering 200 ears, one finds a total of 42 with tympanic membranes ruptured, or 21 per cent of all ears involved.

Colledge [4] found that in blast injuries Shrapnell's membrane never ruptured. No perforations were found in Shrapnell's area in my patients. The most common site of perforation was found by Negus [5] and Palmer [6] to be the anterior-inferior quadrant. Alexander [7] found this

TABLE 5.—*Location of the Perforations*

	Number	Percentage
Central	18	43
Anterior-inferior quadrant	14	33
Posterior-inferior quadrant	7	17
Anterior-superior quadrant	1	2
Multiple perforations	2	5
Total	42	100

TABLE 6.—*Healing of the Perforations*

	Unilateral	Bilateral	Total	Percentage
Healed	23	6	35	83
Unhealed	5	1	7	17
Total	28	7	42	100

lesion more frequently in the anterior than in the posterior quadrant. The central perforations (43 per cent) and those in the anterior-inferior quadrant (33 per cent) were the most frequent in this series. In 2 cases multiple perforations were found (table 5).

The prognosis for healing of the perforation is good. Eighty-three per cent of the perforations healed (table 6). The majority did so in less than one month from the time of injury.

4. Colledge, L., in Discussion on Injuries of the Ear, Proc. Roy. Soc. Med. **34**:5-18 (Nov.) 1940.

5. Negus, V. E., in Discussion on Injuries of the Ear, Proc. Roy. Soc. Med. **34**:5-18 (Nov.) 1940.

6. Palmer, G. W.: Blast Perforation of Ear-Drums, Brit. M. J. **2**:233 (Aug. 21) 1943.

7. Alexander, A. B.: Rupture of the Drumhead as a Wartime Injury. Brit. M. J. **2**:195-196 (Aug. 9) 1941.

INTERSTITIAL HEMORRHAGE

Interstitial hemorrhage of the tympanic membrane was found in 6 patients. Watkyn-Thomas [8] expressed the belief that the petechiae seen on the drum are the result of the suction phase of the blast wave. No hemorrhages were observed in the tympanic cavity in the presence of an intact drum.

ACUTE SUPPURATIVE OTITIS MEDIA

According to Collins [2] otitis media occurring after blast injuries results most commonly from the presence of foreign material and organisms blown into the middle ear by the blast. The other etiologic factors found by him were external otitis, faulty treatment and nasopharyngeal infections. Watkyn-Thomas [8] found that recrudescence of old suppurations was exceedingly common after explosions. In Birrell's [9] series all patients in whom otitis media developed gave a history of using "drops" or syringing. Collins [2] found that otitis media developed in 22 per cent of the ears with traumatic perforation of the tympanic membrane. Palmer [6] found infection in 38 per cent, and Alexander,[7] in over 50 per cent.

In this series suppuration of the middle ear occurred in 8 patients, unilaterally in 6 and bilaterally in 2. The total number of ears infected was 10, or 5 per cent of all ears exposed (200). Twenty-four per cent of the ears with ruptured tympanic membranes (42) became infected. All patients in this series had intelligent and proper care of their ears before coming under the observation of a specialist.

ADDITIONAL FINDINGS

Tinnitus.—A majority of the patients complained of tinnitus. In many it was the predominating symptom. The tinnitus was frequently persistent and extremely annoying.

Labyrinthine Irritation.—Suggit [10] found that only 1 of 69 patients showed signs of labyrinthine irritation. Barrow and Rhoades [11] found no signs of labyrinthine involvement in 200 patients with blast injuries. Alexander [7] expressed the opinion that the vertigo occurring after a blast injury with rupture of the tympanic membrane was not due to damage of the inner ear but was due to irritation caused by cold air. Vertigo was a severe complaint in 1 patient in this series.

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8. Watkyn-Thomas, F. W., in Discussion on Injuries of the Ear, Proc. Roy. Soc. Med. **34**:5-18 (Nov.) 1940.

9. Birrell, J. F.: Comparative Report on Ear, Nose and Throat Work in the Army at Home and Overseas, J. Laryng. & Otol. **59**:16-31 (Jan.) 1944.

10. Suggit, S.: Deafness Resulting from Gunfire and Explosions, J. Laryng. & Otol. **58**:313-326 (Aug.) 1943.

11. Barrow, D. W., and Rhoades, H. T.: Blast Concussion Injury, J. A. M. A. **125**:900-902 (July 29) 1944.

Headache.—Often this was localized to the side of the injured ear, and it was a frequent complaint. The syndrome of demonstrable deafness, headache and tinnitus was encountered frequently. In the case of one who has been exposed to high explosive blasts, this syndrome is pathognomonic not only of blast injury of the ear but also, in my opinion, of cerebral concussion.

Tuning Fork Tests.—In all patients the Rinne test showed that air conduction was better than bone conduction. Where no gross injury had been done to the structures of the middle ear, the Weber test always showed that the sound of the vibrating tuning fork was referred to the better ear.

Suggit,[10] Silcox and Schenk [12] and Colledge [4] expressed the opinion that rupture of the tympanic membrane protects the cochlea from the blast and that the degree of deafness is usually greater when the tympanic membrane remains intact. Collins [2] contended that rupture does not play any part in determining the absence or the severity of the deafness. In this series all extremes of deafness have been observed both with and without rupture of the tympanic membrane. No well defined correlation was demonstrated between the cochlear damage and the rupture of the tympanic membrane. It is my impression that rupture of the tympanic membrane plays an insignificant role in determining the severity of deafness resulting from blasts.

TREATMENT

Prophylaxis.—No satisfactory mechanical method of protecting the ears against blast injuries was in common use in World War II. None of the patients reported here used any means of protection. The reasons given for this neglect were summed up by Guild,[13] who stated that (1) dry cotton is ineffective in stopping even moderately intense detonation waves and that (2) cotton soaked with oil or petrolatum and solid obturators are entirely unsuitable for use by those whose duties require that they be able to hear ordinary sounds well at all times.

In the active treatment of these patients, it is essential that they be immediately evacuated from the front and that they should not be exposed to any further firing. There is discouragingly little that the otologist can accomplish in treating patients for deafness uncomplicated by rupture of the tympanic membrane and aural suppuration. Whatever improvement of hearing occurs does so without his aid in the uncomplicated cases. The maximum improvement usually occurs within the first month. After that time, any improvement found can be largely attributed to disappearance or lessening of the annoying tinnitus which so frequently accompanies the deafness.

12. Silcox, L. E., and Schenk, H. P.: Blast Injury of Ears, Arch. Otolaryng. **39**:413-420 (May) 1944.

13. Guild, S. R.: War Deafness and Its Prevention, Ann. Otol., Rhin. & Laryng. **50**:70-76 (March) 1941.

The most important contribution the otologist can make is to institute as early as possible a program of aural rehabilitation for the patients whose deafness is incapacitating. These men are depressed, anxious and introspective as a result of having passed suddenly from a normal hearing world to one where they are partially or completely cut off from sound and the voices around them. Many are disturbed by the annoying tinnitus. My program was to segregate as many of these patients as possible in the same ward and protect them from ridicule and embarrassment. They were encouraged to engage in ward and hospital activities. As soon as possible, a personal interview was held with each patient, and the nature of his disability was explained to him. He was informed about our program to help him and what he could do for himself. In our hospital it was our good fortune to have a capable lip-reading teacher on the Red Cross staff. All patients who could benefit from training in lip-reading were encouraged to attend the lip-reading classes which she taught. The adjustment made by many of these patients was gratifying. It was an excellent start toward the final rehabilitation that was to be achieved in one of the aural rehabilitation centers of the Zone of the Interior.

Tinnitus.—Inflation of the eustachian tube did not influence the tinnitus. No specific therapy was used in treating patients for this symptom.

Rupture of the Tympanic Membrane.—Most of the smaller perforations healed without intervention. Occasionally it was necessary to stimulate the margins of the perforation by mild scarifying or by applying 10 per cent silver nitrate to the margins of the perforation to stimulate healing. Marginal perforations appeared to close more slowly than the others. Large perforations with damage of the ossicles showed no tendency to heal. Most of the perforations that healed did so in less than one month.

Acute Suppurative Otitis Media.—The prophylactic treatment impressed on the medical officers in the forward areas was the preventing of contamination of the ears by absolute avoidance of syringing or of applying "drops" to the ears. Not one of the patients in this series received injudicious aural therapy in the forward areas. The patients themselves had to be instructed not to contaminate their ears. In the presence of a dry traumatic perforation none of the sulfonamide powders was insufflated, because it was felt that there was no advantage to be gained. Many of the patients had other injuries and were treated with sulfadiazine or penicillin or both a short time after being injured.

The active treatment was a daily cleansing of the infected ear with hydrogen peroxide and dry wipes, followed by instillation of one of the following local medicaments: sulfathiazole sodium solution (5 per cent), penicillin solution (containing 1,000 units per cubic centimeter), sulfanil-

amide powder or 2 per cent boric acid in alcohol. The last was used when Bacillus proteus was obtained on culture. Sulfadiazine or penicillin was frequently used systemically, in addition to the local therapy. These infections responded rapidly to treatment. In none of the patients did acute mastoiditis or other complications develop.

SUMMARY

One hundred cases of blast injury of the ears were studied. The important findings in the patients were deafness, rupture of the tympanic membranes, acute suppurative otitis media and tinnitus. High explosive shells most frequently caused the injuries.

Audiometry is essential for accurate study of the deafness of such patients. The characteristic audiometric findings were:

1. A dip, in the audiometric curve at or near the frequency of 4096 double vibrations per second when the hearing loss was minimal.
2. A deepening and broadening of the curve in the frequencies above 2048 when more damage was done to the hearing.
3. A slight depression of the curve in the frequencies below 512 even in the mildest cases.
4. A loss in a wider range, including all the frequencies, more severe in the high tones, but with a tendency toward leveling off of the curve when the deafness was moderate.
5. The least loss almost always in frequencies 512, 1024 and 2048.
6. No hearing of any frequencies of the audiometer when the losses were most severe.

In a majority of the patients (71 per cent) the impairment of hearing was slight.

Tinnitus was often the predominating complaint. The syndrome of headache, tinnitus and demonstrable deafness was frequently found.

Rupture of the tympanic membrane is not an important factor in determining the degree of cochlear damage.

In uncomplicated cases of deafness the maximum recovery usually occurred within one month after the injury. The early institution of a program of rehabilitation was the most important phase of treatment.

Rupture of the tympanic membrane occurred in 21 per cent of the ears involved. The most frequent sites of the perforations were central and anterior-inferior. Eighty-three per cent of the ruptured tympanic membranes healed, and the majority did so in less than one month.

Acute suppurative otitis media occurred in 5 per cent of all the ears exposed and in 24 per cent of the ears in which the tympanic membrane was ruptured. These soldiers had all received proper treatment in the forward areas. The aural suppuration responded rapidly to therapy, and no complications occurred.

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A GENERAL REVIEW OF DR. GEORGE E. SHAMBAUGH'S CONTRIBUTION TO OTOLOGY

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DR. SHAMBAUGH in 1900, after graduation from the medical school of the University of Pennsylvania and two years postgraduate study in Berlin and Vienna, was appointed instructor in the department of anatomy at the University of Chicago and in the department of otology at Rush Medical College. Then began his first important research, which culminated in many papers on the vascular supply of the internal ear. At that time the University of Chicago was becoming recognized as a school in which investigation was fostered in all departments, under the dynamic Prof. William Rainey Harper, who had attracted to the school the best procurable scientists and teachers of various sciences, irrespective of expense. It was essentially a teaching institution; but teaching with inspiration can best come from investigators or laboratories where there is progess in scientific research. The department of anatomy was especially fortunate in having at its head Prof. Lewellys Barker, who had surrounded himself with a group of able and enthusiastic young instructors. Dr. Barker was particularly well qualified to stimulate research from his belief that the path along which medicine must travel was one of science combined with clinical studies which kept students aware of the diverse problems presented in the study of disease, training them in the methods of clinical observation and the means of advancing scientific knowledge. This was characteristic of most of Dr. Shambaugh's investigations—the employment of science to explain problems which presented themselves in the clinic. Dr. Barker was a scholar in the best meaning of that term. He possessed a naturally judicial mentality, which showed itself in the numerous departmental meetings where the various research instructors gave reports of their work and where the reports were discussed by investigators from allied departments. At each such gathering Dr. Barker showed a wise pooling of results and emphasized material which otherwise might have been overlooked.

The investigation of the vascular supply of the internal ear begun by Dr. Shambaugh was not an occurrence of chance. When he identified himself with the department of anatomy he found himself in association with a group of investigators whose field of study was the

distribution of blood vessels in various organs, the liver, the kidney and others, and the ultimate connections of their capillaries. These studies had resulted in a technic whereby the finest veins and arteries could be injected without rupture, the obscuring tissue removed by dissection, by digestion or similar means, and their variations due to advancing ossification evaluated. Finally the whole block could be cleared by various methods so that the relation and the connection of arteries and veins were rendered capable of a clear study. This work was carried on in a congenital environment of friendly and intimate relationships, with criticism and applause, typical of an assembly of young enthusiasts guided by a broad-minded leader. New fields of research were explored, some disappointing and some productive. After months of laboratory work and careful study of each step to avoid errors, Dr. Shambaugh in 1903 issued a monograph in the decennial publication of the University, dealing with the distribution of the blood vessels of the labyrinth of the pig. Later publications extended the investigation to the calf, the sheep and the dog. Such human material as was available was also fully used. Here may be noted the close affiliation that was achieved between the department of anatomy and the stockyards of Chicago and the willingness with which the corporations readily granted material at all stages of development, from embryo to adult life, to aid research.

At that time the descriptions of the vascular supply in the textbooks were confused, and in many details, erroneous. As might be expected, variations were seen in the capillaries during development when the supporting bone around affected the capillaries' growth. It was well known that the blood vessels supplying the structures about the labyrinth communicate freely with those found in its bony capsule: The blood vessels of the mucosa lining the tympanum and branches extend into the wall of the promontory; blood vessels from the dura penetrate the area between the semicircular canals of the fossa subarcuata, and the blood vessels of the temporal bone surrounding the labyrinth communicate freely with the bones of the capsule. Shambaugh's results showed that the prevailing picture was that of an arterial supply brought to the internal ear through a single vessel, a branch of the basilar artery which enters the labyrinth along with the auditory nerve through the internal auditory meatus. The venous blood is carried away from the labyrinth by one vein, the vein of the aquaeductus cochleae, which drains blood from the entire labyrinth. No vein draining blood from the labyrinth was found in the internal meatus, nor one accompanying the aquaeductus vestibuli. Siebenmann, in his work on the blood supply of the human ear, described three channels for the exit of venous blood flowing from the labyrinth. The one, a vein in the internal meatus, drained blood from part of the cochlea.

The second, a vein which left the labyrinth along the aquaeductus cochleae, drained blood not only from the cochlea but from the vestibule as well. The third, a vein which left the labyrinth along the aquaeductus vestibuli, drained blood from the semicircular canals and the vestibule.

One question disputed was whether the ramifications of the labyrinthine artery which supplies the endosteum of the labyrinth penetrate the bony capsule. Politzer believed that he could demonstrate communications between the blood vessels of the mucosa of the cavum tympani and those found in the membranous labyrinth and the endosteum. The opposite view was first expressed by Hyrtl, who held that the blood vessels of the inner ear, from the time the artery enters the labyrinth, form a closed system, and at no point communicate with the blood vessels of the surrounding structure.

An end artery is one which does not anastomose with any other artery. This also applies to its branches. This does not preclude the possibility that its capillaries anastomose with other capillaries. However, such capillary anastomosis is not sufficient to establish a functional anastomosis in time to prevent necrosis of tissue if the end artery becomes occluded. The result will be infarction of the area normally supplied by it. In 1922, in his final paper regarding the blood supply of the internal ear, Shambaugh said, "The arterial supply to the several parts of the labyrinth is through end arteries." In brief, Shambaugh found that all the veins of the labyrinth drained through one channel, the vein of the aquaeductus cochleae. Previous to this work, it had been accepted generally that the veins left the labyrinth by three routes, the vein of the aquaeductus cochleae, the vein of the aquaeductus vestibuli and a third vein in the internal meatus. He succeeded in making a number of excellent injections of the veins of human embryos, as well as of the arteries, but did not succeed in finding in the internal meatus the vein draining the labyrinth. The vein of the aquaeductus vestibuli was constant in the labyrinth of the dog. This picture as described by Dr. Shambaugh was received with approval by anatomists. Its value was quickly recognized by leading otologists, such as Siebenmann, Politzer, Hartman and Gray, all of whom were well qualified by previous investigations to judge. Soon after this work was published, in 1903, an appreciation appeared from the pen of Siebenmann in the *Zeitschrift für Ohrenheilkunde* in 1904.

THE STRIA VASCULARIS [1]

The stria vascularis occupies the outer wall of the cochlear duct. The rich vascular supply indicates an important but then disputed

1. Shambaugh, G. E.: On the Structure and Function of the Epithelium in the Sulcus Spiralis Externus, Arch. Otol. **37**:538-546, 1908.

physiologic function. Regarding this function there were two chief hypotheses: that it was (1) a contractile body capable of putting the basilar membrane on a tension; (2) a secretory body. Retzius believed that there was no evidence justifying the conclusion that it had a contractile function; its anatomic structure indicated that it was a secretory body.

Dr. Shambaugh's careful study showed that these epithelial clumps growing out from the sulcus externus are provided with tubules which open on the free surface, and he expressed the belief that they secrete endolymph; whether these glands secrete all of the constituents of the endolymph or only a part there is no knowing. Dr. Shambaugh's study of the structure of the stria vascularis and his description of the glands within the stria is one of his significant contributions. Today we still believe that the endolymph is secreted by the glands of Shambaugh.

MEMBRANA TECTORIA

Probably Shambaugh's investigation of the structure and function of the membrana tectoria has aroused more controversy among otologists and anatomists than any other of his studies. The heat engendered by the controversy was not conducive to the elucidation of the subject. Nevertheless, Shambaugh's work brought the tectorial membrane into discussion and has contributed to our concepts of auditory function. It was the work of Shambaugh and Hardesty that brought it into the limelight.

In 1891 Ayers presented the first suggestion that the tectorial membrane might be stimulated by sound waves. He was followed by Rutherford in 1898 and Kishi in 1907. In 1897 Siebenmann indicated that he believed the tectorial membrane is an efficient structure for receiving the sound waves and transmitting them to the hair cells. In 1907 Shambaugh selected the tectorial membrane as the organ which analyzed the incoming sound waves. He said, "The fundamental principles in the resonator theory of Helmholtz still remain as the most plausible explanation of the various phenomena of tone perception. With the physiological and clinical facts which we possess, we can hardly escape accepting a resonator theory in some form."

The tectorial membrane varies considerably in different microscopic preparations, and it is still an open question whether it preexists in the living state in the form in which it is seen after it has been treated with fixative and prepared for study. There is some reason to believe that it may represent a postmortem coagulation or condensation of a much more diffuse colloidal structure or material in the ductus cochlearis. Structurally it consists of multitudes of delicate fibers of unequal length

(or lamellas—Shambaugh) embedded in a transparent matrix of a soft, semisolid collagenous character with marked adhesiveness. It has been concluded by many anatomists that few of the fibers extend the entire width of the membrane; none are attached at both ends. Its specific gravity is but little greater than that of the fluid in which it lies. Shambaugh held that the tectorial membrane does not float free above the organ of Corti but is attached to the inner supporting cells by its stripe of Hensen and also to the hair of the hair cells. He believed that the membrana tectoria is so constituted anatomically as to be capable of responding to the most delicate impulses passing through the endolymph.

Hardesty's ideas differed in several respects from the conclusions of Shambaugh. He wrote, "In the first place it is considered probable that the tectorial membrane does lie free over the organ of Corti and that the auditory hairs do not project into it. Its structure consists of multitudes of delicate fibers of unequal length, embedded into a transparent matrix of a soft, collagenous, semisolid character with marked adhesiveness. It is very improbable that the membrane is capable of acting as a sympathetic resonator."

The resonance theory of Helmholtz postulates that different portions of the basilar membrane vibrate selectively in response to different frequencies. Whether the tectorial membrane can so act is still debated, and it is argued that the anatomic structure of the tectorial membrane has not the requisite physical structure to act as a resonating body. It may be remarked that corresponding difficulties exist regarding the basilar membrane.

THE WIDESPREAD INCIDENCE OF DEFICIENCY OF HEARING

Many will regard Dr. Shambaugh's most valuable contribution to otology to be his efforts to awaken not only the medical profession but the general public to the widespread incidence of deafness in the population and particularly to the extent of deficiency of hearing in children. In this he was wholeheartedly aided by the members of the American Otological Society and encouraged by the National Research Council. He bent all his efforts through patient systematic examination of thousands of cases, with the assistance of members of the staff of the department of otology of Rush Medical College, to making accurate surveys with a view to future remedial measures.

Through this inspiration and careful work the way has been opened for accurate and early diagnosis of deafness, and his name will be honored among those who have made significant contributions to medical science. Let the final word be that of Dr. Shambaugh. Writing in the ARCHIVES OF OTOLARYNGOLOGY in 1930, Dr. Shambaugh said:

Unless one has undertaken to make a careful functional examination of the hearing and the vestibular reactions in a deaf child he can hardly appreciate the time and effort and, I might add, the almost infinite patience required for completing this investigation of 5,348 children. I cannot praise too highly the conscientious, careful and persevering work of those who participated in these examinations extending over many months. . .

If we have succeeded in throwing new light on these problems of the deaf and have furnished some facts that may stimulate others to further research, leading ultimately to the amelioration of the lot of these unfortunate children, we feel repaid for our efforts.

CALCIUM AND PHOSPHORUS, AND PHOSPHATASE ACTIVITY, IN OTOSCLEROSIS

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OTOSCLEROSIS (otospongiosis), being largely a disease of bone, one would suspect in this disorder, at least during its active phases, some disturbance of the blood serum calcium-phosphorus content or balance, and maybe of serum phosphatase activity, also. Many attempts have been made to correlate calcium-phosphorus variations with otosclerosis, but no generally accepted conclusions have been reached. No serious attempt has been made with respect to phosphatase activity. There are several reasons for the negative or unimpressive results. One is the fact that the examination is made so long after the onset or an active stage of the disease that abnormal levels of the components of the blood serum have had time to return to near normal by the time the tests are made. Another reason may be that the so-called normal ranges for calcium and phosphorus values permit such large excursions between the normal extremes that in many people really abnormal changes are not recognizable as such. In other words, values near the upper and lower normal limits may not really be normal for some of the persons examined.

A third reason is that the lesion of otosclerosis appears to be limited to the petrous bone, no comparable pathologic change having been detected in other bony structures of the body. Moreover, it is comparatively a small lesion.

A fourth reason is that otosclerosis is characterized by alternating periods of activity and quiescence, and in some decades of life quiescence occurs more frequently than was heretofore realized and much more frequently than activity. After 50 years of age the ratio of active to inactive lesions rapidly declines, only 1 patient out of 3 showing active lesions.[1] In only about one half of the cases of otosclerosis encountered by Guild [2] at autopsy were active lesions seen. His material, of necessity,

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1. Fowler, E. P.: A Report of Progress: Twenty Years Research in Otosclerosis, Progressive Deafness and Correlated Problems, Central Bureau of Research of the American Otological Society, Inc., December 1946.

2. Guild, S. R.: Histologic Otosclerosis, Ann. Otol., Rhin. & Laryng. 53:246, 1944; personal communication to the author.

was obtained from people who had been sick and who died. Their fatal disease may have influenced the activity or the nonactivity of the otosclerotic lesions.

However, purely on a hit or miss basis one would expect a large proportion of the laboratory examinations to have coincided with a period of activity. But, so far as such activity is reflected in blood changes, few impressive correlations have been obtained.

Evidently a modification of the approach to the problem is needed. Guild's statistics suggested one to me. In every instance of ankylosis in Guild's material the lesion showed activity, and in at least a majority of the cases of otosclerosis in which autopsy was done before middle life the process was in an active stage, so that one would expect to obtain some confirmation of calcium change in patients examined during middle life, at least, if in fact a variation does occur during activity of the lesions, irrespective of the exact time of the laboratory examinations. Moreover, there should be some differences between middle life and the older age epochs.

In 1930 I [3] reported the calcium and phosphorus contents of the serums of 58 white persons who had otosclerosis.[4] There was little difference between the variations found in the patients and in the controls (people with no indication of otosclerosis). However, as otosclerosis eventually develops in over 8 per cent of the white population,[2] over 8 per cent of my white controls might be expected to harbor the lesion subclinically. Such a high percentage in the controls would confuse the picture, because in some of the controls otosclerosis would be in an active stage and their blood chemistry might show changes—that is, if such changes were in fact related to the activity of the lesion. It would distort the standard used for comparison by bringing it closer to the levels shown in the patients with otosclerosis. In my 1930 report the number of subjects was too small (both otosclerotic persons [58] and controls) to warrant attributing the variations to changes in the activity of the lesion or to exclude the effect of some other factor modifying the data. In other words, even if in fact there is a blood calcium deficiency during activity or progress of the lesion, many of the apparently normal persons (controls) would show measurements similar to those of the abnormal (otosclerotic) persons.

The lesion of otosclerosis appears to be self perpetuating. This may be because once it is established, less change is required in some or all of the etiologic factors to reactivate it, than was needed to establish or to activate it at its inception (may be vice versa). Certainly no clear

3. Fowler, E. P.: Calcium, Phosphorus and Cholesterol in Otosclerosis, Arch. Otolaryng. **13**:77 (Jan.) 1931.

4. The laboratory determinations were under the supervision of Dr. Andrew A. Eggston.

causes have been discovered to account for its perpetuation. This makes it difficult to detect blood serum variations by any of the methods now available.

Be this as it may, I do not believe it is self perpetuating, and I have persisted in my chemical analyses of the blood and now report results obtained in studies of 160 additional patients (130 white adult females and 30 males). I report also the results of determinations of the alkaline phosphatase. [5]

One cannot ignore the classic observation that deafness due to otosclerosis appears to develop or increase in women more frequently at or near the time of puberty, of pregnancy, of lactation or of the menopause—periods when even normally more profound changes occur in the endocrine functions and in mineral metabolism than at other periods of life.

Pertinent, therefore, is the observation that the incidence of otosclerosis, as well as the incidence of activity of the lesion found histologically, is lowest in white females coming to the autopsy table before 11 years of age (before puberty); also, the observation that it diminishes rapidly after 50 years of age (after the menopause).[2]

In mothers a high incidence of otosclerosis coincides with periods of increased need for calcium and phosphorus to supply the baby in the womb and the nursing infant. There is an increased need of calcium and phosphorus in the rapidly maturing child. Other minerals are also necessary for the proper formation, development and growth of bony tissues at such times. In other periods of life and especially in old age less calcium and phosphorus are required. During gestation and lactation, if the mother is deficient in calcium and phosphorus, not only the child but the mother herself is adversely affected.

Calcium and phosphorus normally complement one another. The levels vary greatly in different animals, depending in part on the amount and the rate of the bone or egg shell formation. Also there are many exceptions to the rule; for instance, in the human subject normal levels of phosphatase, calcium and inorganic phosphate may obtain even in the presence of menopausal osteoporosis, a condition which may represent an exaggerated postmenopausal or emotional process in the body as a whole.

It is well known that parathyroid preparations will mobilize and pull calcium from the bones, but it is not so well known that estrogens may act in this way. Both the estrogenic and the parathyroid hormone show changed activity at the critical periods of life mentioned in a foregoing paragraph. In women these hormones usually vary with changing activity of the thyroid and other endocrine glands. Either in

5. The chemical studies of the blood were under the supervision of Dr. Oliver S. Hillman.

excess has the power of absorbing the calcium. Excessive estrogen may resorb bone and redeposit it as endosteal (medullary) bone.[6]

In my material, among white males the serum phosphorus was below 3.5 mg. per hundred cubic centimeters (the normal limit) in 56 per cent, and among white females, in 62 per cent (chart). In 80 per cent of the males the calcium was below 9.5 mg., and in 20 per cent it was below 9 mg., per hundred cubic centimeters. Among the females, in 66 per cent calcium was below 9.5 mg., and in 85 per cent it was below 10 mg., per hundred cubic centimeters, the average normal. Only in 9 was it below 9 mg., and in 17 it was exactly 9 mg. (chart). In the total material (males and females) calcium was below 9.5 mg. in 70 per cent. Of the controls, both children and adults, only 47 per cent had calcium levels below 9.5 mg. (people with uncomplicated nerve deafness).

It will be noted on the chart that low phosphorus is frequently associated with low calcium. As a matter of fact, phosphorus was low in 62 per cent of the persons showing low calcium, and calcium was low in 68 per cent of the persons showing low phosphorus. This means that in about two thirds of the persons with otosclerosis showing calcium below 9.5 mg. the phosphorus was below 3.5 mg.; also that in over two thirds of the persons showing phosphorus below 3.5 mg. the calcium was below 9.5 mg. This association of low phosphorus and low calcium in otosclerosis has, as far as I am aware, not heretofore been demonstrated.

Do the tendencies toward low values mean anything, and if so what? In my opinion they do mean something, but I am loath to say exactly what, except that they should not be ignored. Low normal values may not affect the normal person, but in those with hereditary tendencies toward otosclerosis they may be the determining factor in projecting the disease into activity.

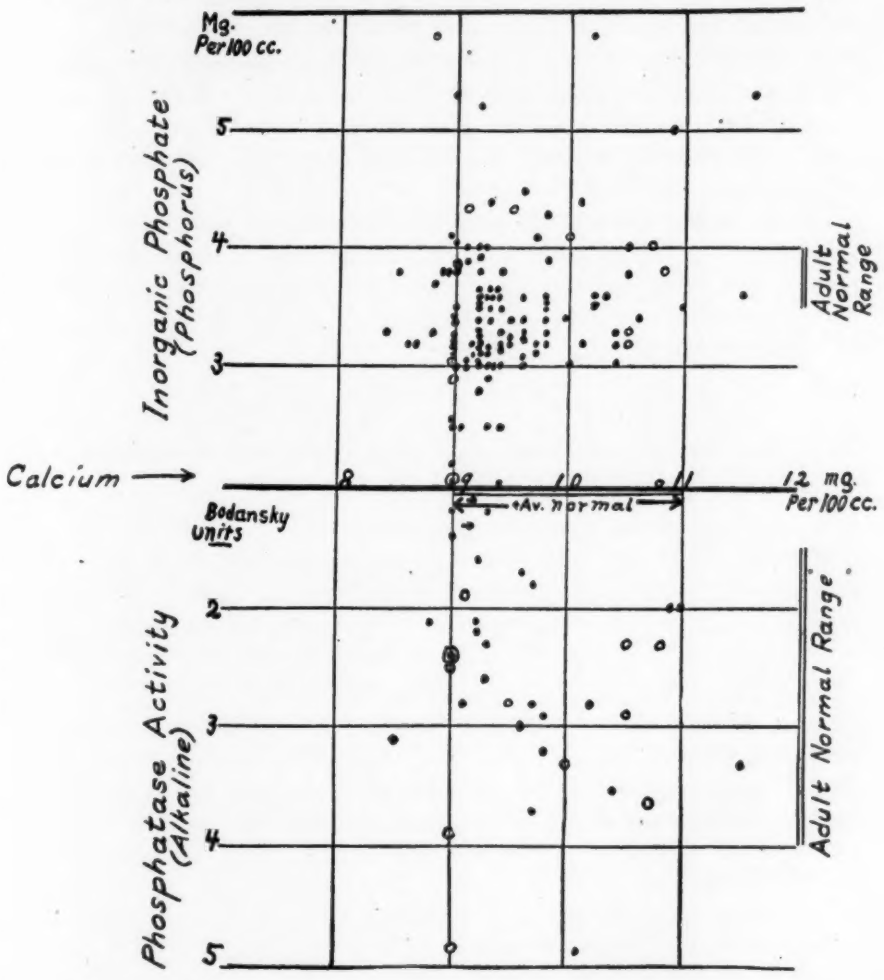
Repeated examinations at intervals of several years showed in my patients no impressive change in the calcium and phosphorus contents of the serum, except for those patients who followed my advice and were faithful in taking extra amounts of one or both as indicated, and those who because of a more properly balanced diet obtained sufficient amounts of these substances to raise the values. In these patients there appeared to be a stabilization of the deafness and therefore, by inference, a deterrent effect on the activity of the lesion of otosclerosis.

SERUM PHOSPHATASE ACTIVITY

Phosphatase, like other enzymes, is revealed by its activity. There are many phosphatases, classified as so-called alkaline and acid phosphatase. The former act optimally at a distinctly alkaline p_H; the latter, at a distinctly acid p_H.

6. Riddle, O.: Personal communication to the author; see also Physiol. Rev. **23**:139, 1943.

Alkaline phosphatase activity is more widely distributed in the tissues than acid phosphatase activity. High concentrations of alkaline phosphatase are found in the liver, the kidneys, intestinal mucosa, bone, ossifying cartilage and leukocytes. Its activity is found in relatively smaller amounts in the blood serum, but in far greater amounts than is acid phosphatase activity. In addition to playing some role in the process



The upper scatter graph shows the values of serum phosphorus (inorganic phosphate) and serum calcium determined in chemical examinations of the blood of 140 white female patients with otosclerosis (uncomplicated in 127, represented on chart by black dots, and complicated with nerve deafness in 13, represented by circles).

The lower scatter graph shows the values of alkaline phosphatase found by study of phosphatase activity in the same group.

The double lines indicate the average normal range of values for adults.

by which glucose is absorbed through the intestinal wall and resorbed in the renal tubules, it appears to be a necessary factor in the formation of osseous tissue. In disease an elevation of this enzyme is more important and more common than a drop. It is the alkaline phosphatase activity with which I am here concerned.

Under specific conditions the normal serum phosphatase range is calculated to be 4 to 14 Bodansky[7] units for infants and children and usually higher for infants below 5 months of age. In children values even near the upper limit of the normal range should arouse attention. For adults the range is from 2. to 3.5 (some say 1.5 to 4) Bodansky units.

As shown in the chart, there was a tendency toward low values in women whose blood calcium was low or low normal. No high levels were revealed.

Phosphatase values rarely vary much, even over many months, in particular cases. High values must be considered and interpreted in connection with other results of the chemical examination of the blood. The bodily conditions associated with high and low levels of serum alkaline phosphatase may be listed as follows:

Values tend to be higher with:	*Values tend to be lower with:*
Hyperparathyroidism—primary and secondary. The less elevated the values the quicker the return to normal following correction of the condition. (A return to normal indicates that recalcification of the skeleton is complete.)	Hypoparathyroidism (surgical)
Pronounced leukocytosis (values are slightly higher)	Cachectic states, even those resulting from cancerous growth if this has not metastasized to the liver or to the skeleton
Various disorders of the liver and biliary passages	Celiac disease
Paget's disease (osteitis deformans), unless healing. (Calcium and phosphorus are usually normal unless the disease is associated with hyperparathyroidism.)	Chronic nephritis with destruction of renal tissues
Active rickets of infancy and early childhood (close correlation). (Phosphatase is a more reliable index than inorganic phosphorus; it is much more reliable than serum calcium, which tends to be low.)	Idiopathic and renal dwarfism
Osteomalacia (bone softening). (Calcium and inorganic phosphorus values are low.)	Cretinism

7. Bodansky, A.: Determination of Plasma Phosphatase, Proc. Soc. Exper. Biol. & Med. **28**:760, 1931; Phosphatase Studies, J. Biol. Chem. **101**:93, 1933.

Values tend to be higher with:	*Values tend to be lower with:*
Sprue (nontropical with osteoporosis)	Cancerous growth if the liver or the skeleton has not been involved
Osteoporosis if this is not reactive. (If osteoporosis is senile or menopausal, the phosphatase, calcium and inorganic phosphate levels may all remain within normal limits.)	Otosclerosis with low serum calcium (Fowler)
Osteoplastic tumors and metastases (osteogenic sarcoma)	(Low values are found after recalcification processes.)
Osteitis fibrosa cystica (generalized)	(Exostoses, enchondromatosis and achondroplasia appear to cause no elevations.)
Fibrous dysplasia of bone	(Occasionally low values may be found for normal adults.)
Osteopetrosis (marble bone)	
Osteogenesis imperfecta (if fracture is present)	

The principal factor underlying elevations of serum phosphatase appears to be abnormal formation of new osseous tissue (reactive osteogenesis).

The principal factor underlying decline of serum phosphatase appears to be the completion of processes of recalcification. Normal values indicate that recalcification of the body is complete and that the disease is cured or quiescent. It was thought that this might also obtain in inactive otosclerosis and that increased values might be found in active otosclerosis. The difficulty is that skeletal involvement must be considerable to elevate phosphatase values, and in otosclerosis there is no large skeletal involvement. This may account for the small number of variations in the cases here reported. Little or nothing having been uncovered by the determinations of phosphatase levels so far as otosclerosis is concerned, they will no longer be routinely included in the laboratory examinations of my adult patients unless some good reason is found for doing so.

SUMMARY

The blood serum calcium, phosphorus and phosphatase determinations reported here indicate that phosphorus (inorganic phosphate) was below 3.5 mg. per hundred cubic centimeters (the low limit) in 62 per cent of the women and 56 per cent of the men. The calcium also was definitely on the low side, being below 9.5 mg. per hundred cubic centimeters in 70 per cent (66 per cent of the women and 80 per cent of the men). The phosphorus was below 3.5 mg. in two thirds of those with calcium below 9.5 mg., and the calcium was below 9.5 mg. in over two

thirds of those with phosphorus below 3.5 mg. The percentage of patients in which association of low calcium and low phosphorus occurred has not heretofore been demonstrated in studies of otosclerosis.

The phosphatase activity was within normal limits except in a few of those with low calcium. The possible significance of such findings is discussed.

It would appear that the important implication of these blood chemistry determinations is that intake and utilization of calcium and phosphorus should be carefully studied and controlled, especially during those periods of life when an ample supply is necessary for the good health of the body as a whole, and particularly in those cases in which there is a family history of otosclerosis, with a view to insuring the maximum required for health of the bone containing the inner ear. In view of the findings it would be folly to neglect this in spite of the meagerness of knowledge of the causes of the lesions of otosclerosis or of the causes of their activity.

140 East Fifty-Fourth Street.

ABSTRACT OF DISCUSSION

DR. T. E. WALSH, St. Louis: Dr. Fowler has confirmed the findings of previous investigators that in patients with otosclerosis the serum calcium and phosphorus levels are definitely within the accepted limits of normal; that is, between 9 and 11 mg. per hundred cubic centimeters for calcium and between 3 and 5 mg. for phosphorus. In the scatter graph which he showed, he finds that more of his otosclerotic patients had calcium values between 9 and 10 than between 10 and 11 mg., and similarly more had phosphorus values between 3 and 4 than between 4 and 5 mg. He attaches some significance to this finding. I suggest, however, that before too much emphasis is placed on the "low normal" finding, one should be careful to rule out possibilities of error in the technic of the determinations. For example, what were the plasma protein values? It is well known that serum calcium values are low in hypoproteinemia. Further, how do these values for otosclerotic patients compare with values for a similar number of people in the same age group, the determinations being done by the same observer? Dr. Fowler seems unwilling to accept these calcium and phosphorus values as normal in spite of the fact that they fall within the accepted normal range. He postulates that in many cases the determinations may have been done in a period of quiescence of the disease. He further states that in all of Guild's cases in which there was ankylosis of the stapes the disease was found to be active. It has been my experience that in more than 90 per cent of 170 patients with otosclerosis who were operated on the stapes was fixed. Day has also emphasized this point. Therefore, one must assume that in all of these patients the disease was active. I presume my criteria for clinical otosclerosis and Dr. Fowler's are similar; so it seems that in most of the cases the otosclerosis possible of diagnosis is in an active state and that, therefore, if the calcium and phosphorus values are significant, they should be more uniform.

Dr. Fowler states that the incidence of activity of the lesion found histologically is lowest in white girls below the age of 11 years and that it diminishes rapidly after 50 years of age. He further cites the fact that in mothers the incidence of otosclerosis coincides with periods of increased need of calcium for supplying the baby in the womb and the nursing infant. He throws particular significance on

this in an effort to emphasize the importance of calcium and phosphorus values in otosclerosis. However, it appears that he is arguing against the factual evidence that he has produced. One might just as easily argue thus: During pregnancy and parturition there is an alteration of the endocrine functions in women. This alteration may also occur at puberty. Consequently one should expect an alteration in the basal metabolic rate. Whether it is altered or not the basal rate is of significance in the diagnosis and treatment of otosclerosis. I believe it is safer to treat factual evidence objectively than to try to make the evidence fit a theory.

Dr. Fowler has treated certain patients with a dietary regimen increasing their calcium and phosphorus intake. He states that in those who followed his instructions he has been able to raise the serum calcium and phosphorus levels and that in such patients there appeared to be a stabilization of the deafness. If this can be shown to occur with any regularity over a sufficient length of time, it is indeed a most significant observation, and assuredly treatment along these lines should be tried in early cases of otosclerosis. It should be remembered, however, that in otosclerosis the hearing does not deteriorate at an even pace but may remain at one level for years and suddenly become much worse and again remain steady for years more. This step by step deterioration of the hearing I believe to be characteristic of the disease. If medical therapy along the lines of diet and phosphorus-calcium increases is started (and I firmly believe it should be if it offers even the remotest chance of success), it should be carried on only to the extent to which it is of value, and most careful observations of the hearing should be made over a long period. I believe that excessive zeal in medical therapy may deny relief of deafness to those in whom surgical treatment would offer a good chance of success. There are, of course, two schools of thought on this: those who are enthusiastic about the chances of the fenestration operation and those who are still skeptical in this regard. Let me make a plea for more openmindedness on both sides. Even if one differs with the views of Dr. Fowler, the arguments which his paper provokes will stimulate further thought and investigation.

Dr. De Forest C. Jarvis, Barre, Vt.: Because the drinking water in my part of Vermont is loaded with calcium, the problem of calcium metabolism has been a constant one. Twelve years ago I sought the aid of a nationally known research dentist who does a blood calcium and a blood phosphorus determination for every patient. I presented the significance of changes in the color of the mucous membrane of the nose and suggested that a relationship might exist between the color changes and the blood phosphorus and blood calcium levels. He wrote me that he was interested and would add a study of the color of the mucous membrane of the nose to his examination routine. In due time he reported that increased redness of the mucous membrane of the nose was associated with high blood phosphorus and low blood calcium. On the other hand, pallor of the mucous membrane was associated with low blood phosphorus and high blood calcium. His published work shows that sugar, molasses, honey, alcoholic beverages and insulin will lower blood phosphorus and raise blood calcium. Kelp from the ocean will balance phosphorus and calcium in the blood and bring about their assimilation. As a result of observations which I have made I have learned that if one wishes to influence the deposit of calcium in places natural for its deposit in the body and influence its removal in unnatural places of deposit this may be accomplished by giving attention to the daily intake of food and prescribing iodine, potassium and acidulation.

Dr. Edmund P. Fowler Sr., New York: In the main I agree with Dr. Walsh and thank him for his discussion. As for errors, the determinations were done by

the same laboratory, and the controls were deafened people without otosclerosis. They were all deafened people. The controls had no low tone losses and therefore no deafness from otosclerosis.

As for the lesion being active in all instances of ankylosis, this does not necessarily follow, in spite of the fact that in Guild's material all the cases of ankylosis did show activity. Eventually at some period the ankylosis will come to arrest and the lesion will become inactive in the same way that the processes in other regions of the capsule do.

In all of my cases the basal metabolic rate was determined, but I have not reported on this. Also, the cholesterol was determined, and many other things. I thought that it was better to limit the paper to the subjects discussed.

In regard to the stability following the treatment: The treatment usually was largely advice, and little advice was necessary if calcium and phosphorus were near the normal. My own personal view is that I see fewer people with increasing deafness than I did before. This does not mean that the course recommended is a cure for otosclerosis. There is none.

In regard to Dr. Jarvis' discussion, the difficulty is that in otosclerosis the calcium is not being deposited in unnatural places. It not only is being deposited in natural places but is being taken away first and then put back.

Some may laugh at the fact that he gives vinegar, molasses, etc., but there is much sound sense back of that. If anybody can do anything, or even tries to do anything, for the better understanding of calcium metabolism, I welcome it.

Case Reports

OTOLARYNGOLOGIC MANIFESTATIONS OF THROMBOPENIC PURPURA

SAMUEL L. FOX, M.D.
AND
G. BROOKS WEST Jr., M.D.
BALTIMORE

THOUGH epistaxis is a common occurrence in childhood, it is seldom of severe nature, and therefore its many causes are often overlooked. Among the chief causes of epistaxis occurring in children are presence of a foreign body, instrumentation, congenital syphilis, diphtheria, the acute exanthems, typhoid, rheumatic fever, malaria, septicemia, anemia, leukemia, purpura, scurvy, tuberculosis, tumors, noncancerous as well as cancerous, and, rarely, hereditary telangiectasia of the nose. In the majority of cases epistaxis, from whatever cause, occurs in the anterior portion of the septum and is usually not difficult to control. Although purpura is not a common cause of epistaxis in children, the case to be reported is interesting from an otolaryngologic standpoint because the presenting symptom and the first signs of the disease were referable to the ear, the nose and the throat.

REPORT OF A CASE

J. S., a 6 year old white boy, was brought to the emergency room of the hospital by his parents at 7 p.m. on March 30, 1947, with the complaint of high fever and nosebleed. The parents stated that the child had been in good health until March 28, when he suffered slight nasal bleeding which soon stopped without medical treatment. On the morning of admission he began to have a "high fever." In the afternoon he had a severe nosebleed for which they sought medical attention. There was no history of previous bleeding from the nose, nor were there any other pertinent data in the past history. The child had had no medication for the past several months.

When the patient was in the emergency department, bleeding was found to be moderate, from both nostrils, and no definite area of bleeding could be found. Consequently, the nose was packed anteriorly with one-half inch iodoform gauze tape and the patient was admitted to the pediatric and otolaryngologic services, for further observation.

On reaching the ward, the child appeared in no acute distress, and the nosebleed had been satisfactorily controlled with packing. The blood pressure was 90 systolic and 50 diastolic; the pulse rate was 100 per minute and the temperature was 103 F. rectally. The general physical examination, made by the intern, gave essentially negative results. During the night the patient removed his own nasal packing, and only slight bleeding resulted, which soon ceased spontaneously.

Otolaryngologic examination, six hours later, revealed the nasal mucous membranes to be congested and diffusely blushing. The septum was essentially in

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the midline, and there was some blood-tinged mucoid discharge along the floor of the nose on each side. Several dried blood crusts were noted in each vestibule. The teeth were in fair condition. The buccal and gingival mucous membranes were normal. The tonsils were hypertrophied and the postpharyngeal mucous membrane was somewhat congested. The nasopharynx was not examined. Several cervical glands were noted at the angles of the jaw. Examination of the ears revealed the right canal to be normal and the right tympanic membrane to show slight congestion posteriorly. The remainder of this membrane was of normal color and the position was good. The left canal was normal, and the left tympanic membrane showed several discrete small hemorrhagic purplish areas. These appeared to be between the layers of the membrane. The color of the remainder of the tympanic membrane was normal, and its position was good. A search of skin, mucous membranes and conjunctiva, failed to reveal any hemorrhagic areas. Examination of the ocular fundi revealed no abnormality. The spleen was not palpable. The heart, the lungs and the abdomen seemed normal. The impression at this time was some hemorrhagic disease, or possibly early acute rheumatic fever.

Laboratory reports gave the hemoglobin content as 77 per cent and the red blood cell count as 3,820,000. The white cell count was 7,000, with neutrophils 62 per cent, lymphocytes 37 per cent and monocytes 1 per cent. The platelet count was 198,000, and the bleeding time was four and one-half minutes, while the coagulation time was three minutes. An undiluted prothrombin time was reported as thirteen seconds.

Electrocardiographic tracings failed to reveal any evidence of rheumatic carditis. The tourniquet test (Rumpel-Leede phenomenon) was positive. Daily follow-up examinations revealed several other purpuric areas on the mucous membranes of the mouth.

The drum membrane completely cleared, the temperature fell to normal, and there were no visible evidences of hemorrhagic disease after forty-eight hours. When the patient was last seen in the outpatient clinic, the mother reported that there had been no further bleeding, and there were no purpuric spots visible. The platelet count was 45,650.

In reviewing the case, the diagnosis of thrombopenic purpura was justified. The nosebleed was attributed to the underlying purpura, and the hemorrhagic spots on the tympanic membrane were diagnostic of hemorrhagic disease. This case was diagnosed as one of thrombopenic purpura only because a thorough physical examination brought out salient features of this disease. Epistaxis in childhood must therefore be regarded as a symptom which requires thorough investigation.

SUMMARY

A case of epistaxis secondary to thrombopenic purpura is presented.

The usual causes of epistaxis in childhood are reviewed.

A tentative diagnosis of thrombopenic purpura was made by otolaryngologic examination.

1205 St. Paul Street.

STAB WOUND OF ORBIT AND ANTRUM

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CHICAGO

PENETRATING wounds of the orbits and sinuses were common during the war. In civilian life, however, they are infrequent. A case observed by me was sufficiently interesting to merit being reported.

A woman about 35 years of age walked into the admitting room of Michael Reese Hospital during the night of April 20, 1947, stating that she had been stabbed "in the eye" during a recent quarrel. She was admitted to the service of Dr. Nathan Crohn, at whose request I later saw her.

Examination on admission revealed the handle of a large pocket knife projecting from the right upper lid, near the internal third. Roentgenograms taken with the knife in situ showed the blade traversing the orbit downward into the antrum, its point reaching the floor near the posterior internal corner at a point close to the posterior border of the hard palate. The knife was extracted, leaving a jagged wound in the lid, which bled moderately.

Examination of the eye on the following day failed to disclose any damage of the bulb, nerve or extrinsic muscles. Examination of the nose also yielded no evidence of trauma except that the antrum was slightly cloudy. There was a moderately reddened, tender area in the right side of the hard palate, near the posterior border, which corresponded to the spot where the point of the knife came to rest.

The patient was treated with penicillin and hot compresses for a few days, and since no untoward symptoms appeared, she was discharged from the hospital one week after her admission.

Examination in the clinic a few days later revealed a mild degree of conjunctivitis and slight edema of the upper lid. The wound was healed.

The case is interesting because of (1) the absence of injury to vital structures, (2) the absence of secondary infection and (3) the presence of the missile in situ when the patient was admitted.

Dr. H. M. Perlmutter, of the staff of Michael Reese Hospital, supplied the photograph of the patient taken on admission.

25 East Washington Street.

Fig. 1.—Patient with a knife plunged through the right orbit into the antrum.

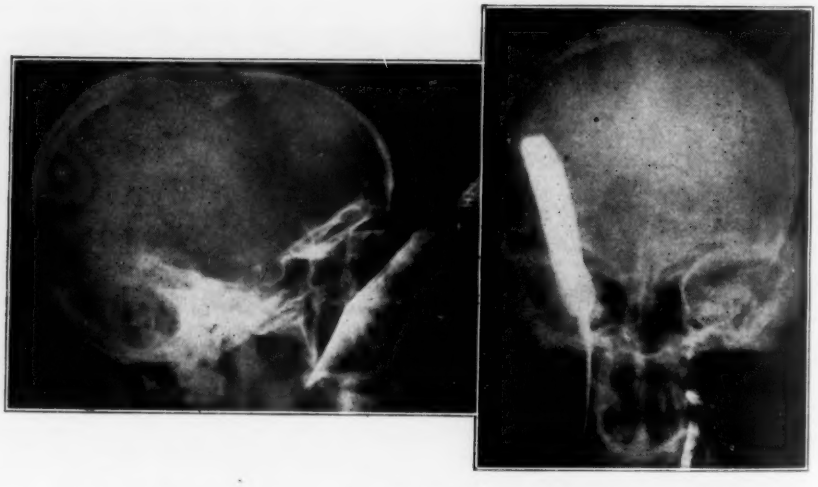

Fig. 2.—Roentgenograms showing the course taken by the blade.

Clinical Notes; New Instruments and Technics

INTRALARYNGEAL APPROACH FOR ARYTENOIDECTOMY IN BILATERAL ABDUCTOR PARALYSIS OF THE VOCAL CORDS

A Preliminary Report

WILLIAM C. THORNELL, M.D.
CINCINNATI

ATTENTION to the surgical treatment of bilateral abductor paralysis of the vocal cords was renewed by King [1] in 1939. Originally he conceived the idea of transposing the anterior belly of the omohyoid muscle to the arytenoid cartilage as a method of replacing the function of the paralyzed cricoarytenoideus posticus muscle. The operation described as a method of opening the cords by the use of the transposed muscle actually was a new method of cord displacement. Hoover [2] has reported success with a submucous resection of the vocal cords. Kelly [3] (1940) has described a procedure in which an arytenoidectomy was carried out through a small window in the thyroid cartilage. Orton [4] has described a lateral transthyroid approach (Trotter's) in which removal of the posterior third of the ala of the thyroid cartilage was carried out and then an arytenoidectomy was performed. The internal perichondrium and thyroarytenoideus muscle was then fixed to the external perichondrium and anterior portion of the split sternohyoid and thyrohyoid muscles.

INTRALARYNGEAL APPROACH THROUGH SUSPENSION LARYNGOSCOPE

The removal of the arytenoid cartilage by the intralaryngeal approach is carried out through the Lynch laryngeal suspension apparatus. After the patient has been anesthetized by intravenous injection of "pentothal sodium" and the suspension laryngoscope is in place, further anesthesia is obtained by applying a 5 per cent hydrochloride solution locally over the vocal cords and the aryepiglottic folds. An incision 1 cm. in length is then made over the upper surface of the arytenoid cartilage, extending anterolaterally into the aryepiglottic fold (fig. 1). The superior border of the cartilage is identified and tightly held by a grasping

From the Division of Otolaryngology, College of Medicine, University of Cincinnati.

1. King, B. T.: A New and Function-Restoring Operation for Bilateral Cord Paralysis: Preliminary Report, J. A. M. A. **112**:814-823 (March 4) 1939.

2. Hoover, W. B.: Bilateral Abductor Paralysis: Operative Treatment by Submucous Resection of the Vocal Cords, Arch. Otolaryng. **15**:339 (March) 1932.

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4. Orton, H. B.: Surgical Approach for Arytenoidectomy in Bilateral Abductor Paralysis of the Larynx, Laryngoscope **53**:709-716 (Nov.) 1943.

forceps (fig. 2). Submucous dissection of the arytenoid cartilage is then carried out. The various muscular attachments are separated from the cartilage on its lateral aspect. The superior portion of the arytenoid cartilage is then rotated laterally so as to facilitate the separation of the muscular attachments on the medial aspect of the cartilage. A sharp laryngeal knife is used to carry out this dissection. It is extremely important to avoid tearing or injuring the mucous membrane of the lateral wall of the larynx, as scarring in this area producing a

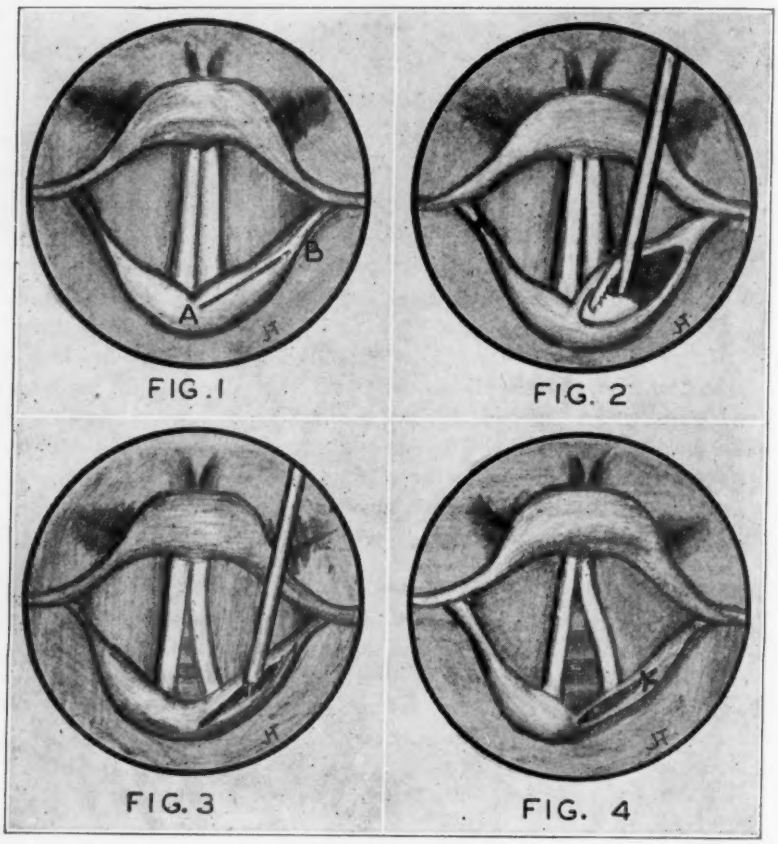

Fig. 1.—Diagrammatic view of the larynx demonstrating bilateral abductor paralysis of the vocal cords. Incision *A* to *B* is made over the upper surface of the arytenoid cartilage and is extended into the aryepiglottic fold.

Fig. 2.—This diagram demonstrates the arytenoid cartilage in position, tightly held by the grasping laryngeal forceps.

Fig. 3.—Diagram showing the electrocautery point entering the space previously occupied by the arytenoid cartilage.

Fig. 4.—This diagram shows the final result that has been obtained when the suture is in place at the anterolateral aspect of the wound.

cicatrix may nullify one's attempt to increase the glottic opening. Following complete removal of the arytenoid cartilage, a guarded electrocautery point is then inserted deeply through the incision along the course of the thyroarytenoid

muscle, beneath the vocal cord, and into the space previously occupied by the arytenoid cartilage (fig. 3). Scarring thus produced by low grade electrocauterization will result in lateral and not mesial contracture. A chromic catgut suture is then used to close the anterior half of the incision, the posterior portion being allowed to remain open to afford drainage (fig. 4).

REPORT OF A CASE

A 54 year old white woman was seen in my office for the first time on Oct. 12, 1945. She stated that she had undergone thyroidectomy in September 1944. Immediately after the operation, respiratory distress developed, which required tracheotomy. Her condition improved, and in four weeks the tracheotomy tube was removed. In March 1945 complete laryngeal obstruction developed, necessitating replacement of the tracheotomy tube, which she had retained up to the time I saw her.

On laryngeal examination, both vocal cords were found to be fixed in the midline position. A tracheotomy tube was in place. On covering the opening of the tracheotomy tube, no air was able to pass through the glottic opening, and both cords remained in their midline position. The remainder of the examination of ears, nose and throat revealed nothing of significance.

On May 4, 1947, the patient entered the hospital, and the following day arytenoidectomy was carried out on the right side through the Lynch suspension apparatus with the patient under anesthesia produced by intravenously given "sodium pentothal." After the suspension apparatus was in position, the cords were painted with a 5 per cent cocaine hydrochloride solution. The procedure was carried out as outlined in the foregoing section. Little bleeding was encountered, and this was completely controlled by the electrocautery unit. Postoperatively pain in the chest delayed the patient's dismissal from the hospital. Physical examination and roentgen studies of the chest revealed no abnormality. On May 14 she was dismissed.

The patient was allowed to use her voice after the third postoperative day. Postoperative laryngeal edema subsided in ten days. The tracheotomy tube was completely sealed off after three weeks and remained sealed until the tracheotomy tube was removed on June 23. The resulting glottic opening as viewed indirectly was 3 mm. wide posteriorly, the right cord being retracted laterally.

On June 28, the patient was again hospitalized, and the tracheotomy opening was closed after application of a local anesthetic. The patient was dismissed from the hospital the following day. Her postoperative course was uneventful.

The patient has no trouble with respiration, and the voice is better than it was before the surgical treatment. The airway is adequate, and it will not be necessary to carry out arytenoidectomy on the opposite side.

SUMMARY

A new and simplified method of approach is presented for the surgical correction of bilateral abductor paralysis of the vocal cord following removal of the thyroid gland. One case is reported in which an intralaryngeal arytenoidectomy was performed through a Lynch suspension apparatus. The procedure outlined is less time consuming and less shocking to the patient than the various external approaches now advocated.

The use of a direct approach for carrying out arytenoidectomy in patients with bilateral abductor paralysis of the vocal cords eliminates the need for a further external surgical operation on the neck. Thus the chances of dangerous infection are minimized. It is realized that, on the basis of this case only, definite conclusions cannot be drawn. Further studies are under way to determine whether such a procedure will be applicable in all cases of bilateral abductor paralysis of the vocal cords.

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Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

OTITIS MEDIA AND COMPLICATIONS

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THE LITERATURE on otitis media appearing in publications of 1946 is here reviewed under headings for ease in comparison of opinions. Some articles are commented on under more than one heading. Comments of the reviewer appear in brackets.

ANATOMY

Seppälä [1] conducted an interesting study of the variations of pneumatization of the temporal bone. Roentgenograms were taken of the mastoid bones of 1,524 recruits of Finland aged 20 and 21 years.

Variations in Pneumatization Noted by Seppälä

	Pneumatization of Mastoid Cells, per Cent	Pneumatization of Zygoma	
		Only at Posterior Root, per Cent	Also at Anterior Root, per Cent
Pneumatization very extensive.....	3.1	18	82
Pneumatization normal extent:			
Evenly distributed.....	21.8	87	13
In some way irregular.....	18.0	84	12
Pneumatization slightly arrested.....	32.2	17	0.5
Pneumatization strongly arrested....	14.9	5	..
No cellular development.....	10.0	..	..

11.1 per cent showed evidence of previous suppurative otitis media.

Chronic otitis media was found in 1.4 per cent of the cases and only in cases with arrested pneumatic development.

The author studied three problems: the frequency of various types of pneumatization; otitis media in various types of pneumatization, and complications of otitis media in various types of pneumatization. His findings are given in the table.

Seppälä operated on 711 ears in 678 patients who had pathologic conditions. He concluded that complications may develop early in

1. Seppälä, A. J.: Variations in the Pneumatic Cell System of the Temporal Bone; Acute Otitis Media and Complications in Varying Types of Pneumatization, Acta oto-laryng., 1946, supp. 64, pp. 1-118.

temporal bones in which there is arrested pneumatic development. Apicitis developed in 8 patients, all of whom had extensive pneumatic development. Labyrinthitis developed in 21 cases, much more commonly in temporal bones with extensive pneumatization. Otogenic general sepsis, 24 cases, was more common in arrested pneumatization. Lateral sinus thrombosis, 35 cases, was more common in arrested pneumatization. Brain abscess developed in 6 cases, in isolated zygomatic cells adjacent to the brain. Meningitis was present in 14 cases, occurring in arrested pneumatization.

[This is an extremely interesting study which should be read by all otologists.]

BACTERIOLOGY

Townrow and Barrie [2] reported a fatal case of actinomycosis of the middle ear complicated by spontaneous severe hemorrhage. The patient was a 64 year old horseman who had had an intermittent foul discharge for fifty-seven years. Profuse intermittent bleeding from the ear began eight days before admission. During operation on the mastoid profuse bleeding occurred from a vascular mass in the middle ear; packing was necessary. Autopsy revealed a mass eroding the roof of the middle ear, which proved to be an old laminated thrombus. Microscopic examination revealed actinomycosis. The actinomycosis was probably a recent infection occurring in chronic otitis media. The literature was reviewed, and only 17 cases of actinomycosis of the middle ear were found.

DIAGNOSIS

Hoch [3] advanced an idea that many of the cases of chronic otitis media previously considered due to infection might be partly due to allergy. He gave as evidence that eosinophil leukocytes in large numbers were found in 31 of 94 cases examined. He described these allergic ears as having viscous mucoid discharge, and being very slow to heal. The usual allergic treatment was advised in these cases. [The idea of an "allergic ear" is new to the reviewer. Possibly the use of pyribenzamine (N′-pyridyl-N′-benzyl-N-dimethyl-ethylenediamine monohydrochloride) or benadryl (beta-dimethylaminoethyl benzohydryl ether hydrochloride) would help in diagnosis and treatment in these cases.]

Fletcher and Windholz [4] describe the advantages of roentgenograms of the mastoid taken in the Mayer position in chronic mastoiditis. No

2. Townrow, V., and Barrie, H. J.: Fatal Actinomyces Infection of the Middle Ear, J. Laryng. & Otol. **60**:329-340 (Aug.) 1945.

3. Hoch, H.: Cytological Studies of Chronic Otitis with Special Regard to Eosinophil Leucocytes, Acta oto-laryng. **34**:317-330 (July) 1946.

4. Fletcher, H. A., and Windholz, F.: The Use of X-Ray in Chronic Mastoid Condition, Laryngoscope **56**:602-615 (Oct.) 1946.

other method reveals the region of the attic and antrum so well. The position has been simplified, and details will be published soon. [Owen [5] has been demonstrating this position for many years at the Los Angeles Midwinter Course. His article gives illustrations and angles which help in the taking and interpretation of these roentgenograms. Pictures of the mastoid in chronic mastoiditis are not complete unless taken in the Mayer position.]

PREVENTION OF OTITIS MEDIA

Weiss and Manheims [6] observed 239 cases of scarlet fever in an epidemic in a naval training station. Seventy-five patients were given penicillin in doses of 20,000 units every three hours for five to seven days. One hundred and twenty-eight patients were given sulfadiazine and 36 received neither. In 25 per cent in the group without prophylaxis otitis media developed. Otitis media developed in 15 per cent of those who took sulfadiazine and in only 2 per cent of those receiving penicillin.

Besides preventing otitis media, the administration of penicillin produced early fading of the rash and lessening of fever, toxemia and pain.

Boies [7] reviewed the literature to date regarding the treatment of the eustachian tube regions with radium or roentgen rays, and reports results of the irradiation of the nasopharyngeal area in 73 patients. In the group of 54 patients with impairment of hearing probably due to nasopharyngeal lymphoid tissue, improvement in hearing was satisfactory in 46. Eight did not improve. Four patients with hearing losses in frequencies higher than 2048 did not improve. Seven patients with hearing loss of 30 decibels or more were not helped. Five patients with nasal obstruction and slight loss of hearing in all frequencies were improved. Three patients with mucoid discharge from chronic perforation of the drum were treated with irradiation of the nasopharynx, with no improvement. No bad effects were seen in the series.

AEROTITIS

Fowler [8] reports results of the use of radium applied to the eustachian tube in aviators. Biopsy specimens were taken from lymphoid tissue

5. Owen, G. R.: A Simplified Method of Producing the Axial View of Mayer in Chronic Mastoiditis and Attic Cholesteatoma, Am. J. Roentgenol. **57**:260-263 (Feb.) 1947.

6. Weiss, J. A., and Manheims, P. J.: Penicillin Prophylaxis Against Acute Otitis Media in Scarlet Fever, U. S. Nav. M. Bull. **46**:1711-1715 (Nov.) 1946.

7. Boies, L. R.: Irradiation of Nasopharyngeal Lymphoid Tissue: An Evaluation, Arch. Otolaryng. **44**:129-140 (Aug.) 1946.

8. Fowler, E. P., Jr.: Irradiation of the Eustachian Tube, Arch. Otolaryng. **43**:1-11 (Jan.) 1946.

near the eustachian tube of a young aviator subject to repeated attacks of aerotitis. The specimens were taken just before treatment and one month later. Definite shrinkage of the lymphoid tissue was shown. Records of 80 airmen were studied. Some of them did not have a full course of treatment and some could not be followed up. Of the 66 who had full treatment and could be followed up, 46 (69 per cent) obtained relief from symptoms. All had been grounded repeatedly. Fowler advises use of radium or radon only in collaboration with a competent radiologist.

[A. S. MacMillan,[9] chief of the radiologic department at the Massachusetts General Hospital, recently stated that there is some danger of roentgen ray burns to the hands of the person handling the radium applicators now in rather general use if many patients are treated. He warned also that the nurse holding the head of nervous children with the applicators in place might receive burns if many cases were handled.].

Haines and Harris[10] reported on aerotitis in submarine personnel. They first reviewed the mechanism of aerotitis and stressed the "locking effect" of increasing air pressure on the eustachian tube.

> Discomfort is noted if pressure increases to 15-30 mm. Above 30 mm. differential pressure there may be pain, tinnitus, bleeding and even vertigo and nausea. A difference of 80-90 mm. is usually above that which the tubal muscles can overcome, whereupon with any increase in pressure, the tube becomes "locked," the walls are pressed so tightly that the muscular reflexes, swallowing, etc., are unable to exert their effect. . . . Babes in airplanes and persons asleep are particularly susceptible.

A total of 6,149 men were examined before and after pressure. In 1,654 (26.9 per cent) of these aerotitis media developed. Of the 732 patients with aerotitis who were given radium treatment, 90 per cent experienced good results.

Kelly and Langheinz[11] (dentists working with the submarine group at New London, Conn.) reported in detail the dental findings of malocclusion and the treatment given, with relation to aerotitis. Their theory, developed after a conference with Dr. Stacy Guild of Johns Hopkins, was that free lymphatic drainage of the middle ear and eustachian tube is interfered with by fascial and muscular tensions due to the abnormal movements of the jaw, and that restoration of normal movements (by treating malocclusion) quickly relieves mucosal congestion of the eustachian tube. In 50 cases in which it was decided that malocclu-

9. MacMillan, A. S.: Personal communication to the author.

10. Haines, H. L., and Harris, J. D.: Aerotitis Media in Submariners, Ann. Otol., Rhin. & Laryng. **60**:347-371 (June) 1946.

11. Kelly, W. J., and Langheinz, H. W.: Dental Treatment for the Prevention of Aerotitis Media, Ann. Otol., Rhin. & Laryng. **55**:13-28 (March) 1946.

sion was present dental treatment was given, and aerotitis was eliminated in 46 of the cases, or 96 per cent. In 20 of these 50 cases the adenoids were described as enormous and covering the opening of the eustachian tubes. Yet without radium or any other treatment of the adenoids further attacks of aerotitis were prevented by correction of malocclusion.

[Aerotitis media is no longer a problem of the flight surgeon alone; with increased airplane travel every otologist must be familiar with its prevention and treatment.]

CHEMOTHERAPY AND BIOTHERAPY IN OTITIS MEDIA

House [12] reviewed 2,189 cases of otitis media seen at the Los Angeles General Hospital in 1935 and a comparative series of 1,137 cases seen in 1943 in which the patients were treated with sulfonamide compounds. The incidence of mastoidectomy in cases of otitis media was reduced by two thirds. Complications were reduced by 20 per cent and the mortality rate by 50 per cent. Before sulfonamide compounds were used there was a 90 per cent mortality from otitic meningitis. With the use of sulfonamide compounds, the mortality was 15 per cent. There are fifteen tables giving duration of symptoms, organisms cultured, etc. [Studies with large numbers of cases such as this help to determine the value of new drugs and procedures.]

DeBlois [13] studied the action of penicillin in 50 cases at the Massachusetts Eye and Ear Infirmary. The study was chiefly on the effects observed in local application in the nose and ears. A great many cultures were made and grown both in aerobic and in anaerobic mediums. In the 50 cases, 30 different organisms were found, two thirds of which were streptococci, staphylococci or pneumococci. "In only a small percentage of the cases studied did the areas treated become sterile, and in an even smaller percentage did they remain sterile for any appreciable time." . . . "The action could better be described as bacteriostatic and bacteriolytic." [It is bactericidal in very large doses.] She adds: "Penicillin does not need to be bactericidal in its action to secure good clinical results . . . the mechanisms of host resistence when supplemented with bacteriostatic doses of penicillin usually suffice to accomplish the destruction of the penicillin-sensitive pathogens." She suggested that penicillin is lethal only to bacteria about to divide and that a treatment schedule might be worked out with periods of intensive treatment alternating with periods of rest during which bacteria will divide and be killed with the next administration of penicillin.

12. House, H. P.: Otitis Media: A Study of Results Obtained Before and After Introduction of the Sulfonamide Compounds, Arch. Otolaryng. **43**:371-378 (April) 1946.

13. DeBlois, E.: Penicillin in the Field of Otolaryngology, Arch. Otolaryng. **44**:174-183 (Aug.) 1946.

Loeb [14] treated 35 patients who had acute and chronic suppurative otitis media with penicillin drops and 52 patients with boric acid-alcohol solution, to try the value of local applications of penicillin. Penicillin was used in a solution containing 1,000 units per cubic centimeter, and dropped into the ear canal three times daily after cleaning. The boric acid-alcohol drops were used in the same way. There were in the group 19 patients with traumatic suppurative otitis media, battle casualties in whom the drums had been ruptured by shell, bomb or mine explosions with resulting infection. In the patients with acute otitis media treated with penicillin drops the condition cleared up in an average of 6.8 days, while in those in the comparative series treated with alcohol and boric acid an average of 17.4 days was required. Similarly, in the 6 patients with acute suppurative otitis following trauma the condition cleared up in 3.7 days, and in the 13 patients treated with boric acid-alcohol drops an average of thirty-eight days was required. On the other hand, in 10 cases of chronic otitis media treatment with penicillin did not lessen the discharge; while in 15 chronic cases treatment with boric-alcohol drops cleared up the condition in an average of fifteen days.

[It has been the usual opinion in the past that in acute otitis media there was little possibility of any medication finding its way through the tiny perforation usually present, especially if pressure was present in the middle ear and pulsating secretion was coming out of the perforation with each heartbeat. It is possible that in the case of large perforations penicillin might reach the infected area and be of value. Further trial is indicated.]

Krauss [15] reported on the local use of penicillin solutions, 1,000 units per cubic centimeter, in 19 cases of chronic otitis media and in 4 cases of acute otitis media. He followed the suggestion of Swanson and Baker and "forced it (the penicillin) into the middle ear with a pneumatic ear speculum, using alternate positive and negative pressure." These treatments were given daily at a clinic, and patients used the same solution at home as nose and ear drops, every three hours. The results "were confusing and difficult to analyze . . . the impression was that penicillin (used locally) was of little value." Used intramuscularly, 10,000 units every three hours gave much more prompt relief in acute otitis media than the sulfonamide compounds. He also reported on the local use of penicillin solution in sinusitis and thought it was of definite value when instilled directly into the sinuses or when used by Proetz displacements.

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14. Loeb, W. J.: Penicillin Ear Drops in the Treatment of Suppurative Otitis Media, Arch. Otolaryng. **43**:586-593 (June) 1946.

15. Krauss, F. H.: Penicillin Therapy in Otorhinolaryngologic Practice, Arch. Otolaryng. **44**:647-661 (Dec.) 1946.

Hutchinson [16] published a review of the American literature, carefully done, with sixty references. He says American workers have had available more penicillin than the English to the date of writing, but larger supplies are now available to the English. He hopes to publish reports on his own cases soon.

Smith [17] reported on the use of penicillin in acute and chronic otitis media. About 100 patients from army personnel were treated. Results in chronic otitis media were disappointing; some ears improved with large doses given intramuscularly, "but the time required did not point to any advantage over other methods of treatment." Response in acute otitis media was "prompt and often dramatic." In only 1 case was it deemed necessary to perform mastoidectomy, and from the pathologic process found it was thought that the patient would have recovered without operation. Four patients with mastoiditis with complications were treated with penicillin. One had petrositis, 1 meningitis and petrositis, 1 extradural abscess and 1 facial paralysis. The hemolytic streptococcus was the active organism in all cases. All patients had received sulfonamide compounds from two to six weeks without effect. In the case of petrositis and meningitis there was a relapse and recurrence of meningeal signs nineteen days after mastoidectomy, and five days after administration of penicillin was stopped symptoms of petrositis developed. The mastoid was reopened and a tract found going into the posterior petrous cells. Administration of penicillin was continued eighteen days. Seven days later a second relapse occurred with nausea, vomiting and drainage from the ear. The mastoid was reopened and penicillin given for forty days. The patient then remained well.

Lierle and Paul [18] gave penicillin by mouth to 174 patients and remarked on the great difficulty of evaluating the effectiveness of this method. The patients treated included 31 with otitis media, 16 with chronic otitis media, 14 with external otitis and 36 with acute pharyngitis and tonsillitis. Blood levels were obtained in adults by giving 100,000 units one hour before meals, at 10:00 p. m. and 2:00 a. m.—a total of 500,000 units for a twenty-four hour period. Fasting appeared to be the most important single factor in obtaining blood levels. Urine levels were measured and without exception were higher than blood levels. Blood levels could be obtained as long as seven hours after a single dose of 100,000 units was taken on a fasting stomach. Fourteen different formulas were used in order to try the effect of various buffers.

16. Hutchinson, C. A.: Penicillin in Otolaryngology, J. Laryng. & Otol. **61**:1-17 (Jan.) 1946.

17. Smith, A. T.: Clinical Use of Penicillin in Infections of the Ears, Nose and Throat, Arch. Otol. **43**:12-16 (Jan.) 1946.

18. Lierle, D. M., and Paul, W. D.: Oral Penicillin in Otolaryngology, Laryngoscope **56**:352-359 (July) 1946.

The most satisfactory one was reported to be a mixture of aluminum hydroxide and cottonseed oil or a tablet of aluminum dihydroxy aminoacetate. [Other workers have reported that no buffer is necessary; the powdered penicillin is dissolved in water and swallowed.]

Fox [19] gives a detailed discussion of the management of acute otitis media and external otitis. He advises the use of the sulfonamide compounds or penicillin or both in every case of acute otitis media. [His statement that sulfonamide has proved more efficacious than penicillin would probably be reversed by him in view of later evidence.]

Singleton [20] reviews anatomy, pathology, bacteriology and treatment of otitis media. He especially warns against the too common practice of the general practitioner of prescribing a dozen tablets of a sulfonamide compound for acute otitis media with instructions to stop taking the drug when the temperature becomes normal.

Weiss [21] used penicillin solution, 1,500 units per cubic centimeter, by blowing it into the tympanic cavity through a eustachian catheter in 3 cases. He thinks it is "of some value as adjunct to usual systemic treatment." The procedure was done in the subacute stage in 2 cases of acute otitis media, and once after mastoidectomy when the wound was slow to heal. [As the author says, further experience is necessary in order to evaluate the method.]

Hitschler and Vastine [22] studied 16 children with 26 chronically suppurating ears at the Pennsylvania School for the Deaf. This was 3 per cent of the student body. Seven cases followed scarlet fever, 3 cases measles, 5 cases probably followed measles, and 1 case started following a cold. The average duration was nine years. Fourteen of the 26 deafened ears had more than a 60 decibel loss of hearing. "Chronic otitis media in itself does not cause more than 50-60 decibels loss at the maximum. Usually the loss is much less than this."

Several treatments were tried. The first was insufflation of sulfathiazole powder. Twenty-three ears received an average of sixteen treatments over a three week period: eighteen improved, eleven became dry, four did not change and one was worse. All of the dry ears had recurrence of discharge at some time. Sulfanilamide powder was then tried with about the same results. Ionization with zinc was tried in 9

19. Fox, S. L.: The Management of the Acute Ear, Laryngoscope **56**:312-319 (June) 1946.

20. Singleton, J. D.: Acute Otitis Media: Its Management by the General Practitioner and Pediatrician, J. Oklahoma M. A. **39**:325-328 (Aug.) 1946.

21. Weiss, J. A.: Transtubal Instillation of Penicillin in Acute Otitis Media, Arch. Otolaryng. **43**:17-18 (Jan.) 1946.

22. Hitschler, W. J., and Vastine, M. F.: A Study of Chronic Suppurative Otitis Media at the Pennsylvania School for the Deaf, Arch. Otolaryng. **43**: 226-242 (March) 1946.

cases; there was no improvement in any of them. At the end of one year, twenty ears still drained, but the majority had less drainage. Most ears had been treated before the patients entered the school. Cultures showed mixed infections. Operation was not done, "partly because of psychic trauma," resulting from such a procedure and also because of "the difficulty of after care." [It would seem that ears that have not responded to careful conservative treatments and continue to drain foul-smelling pus should be entitled to operative treatment, in spite of difficulties.]

MENINGITIS AND ABSCESS OF THE BRAIN

A comprehensive discussion of meningitis secondary to infections of the ear and nasal sinuses was held by the Royal Society of Medicine. Smith, Schiller, Cairns and Macbeth [23] participated, as well as other noted English otologists, neurologists and neurosurgeons. Their article should be read by every otologist. A series of 37 cases of meningitis secondary to infection of the ear and sinuses was discussed. Twenty-one of the cases followed otitic infections. Only one organism was found to be resistant to penicillin, Hemophilus influenzae, which was isolated from an infant of 8 months. The infant recovered after receiving sulfadiazine for seven weeks.

Of 38 cases of pneumococcal meningitis, only 2 were complicated by a brain abscess, while out of 10 streptococcal cases a concomitant abscess was present in 6.

The following precautions were advised in the use of penicillin intrathecally. "Excessive dosage (40,000 units or more at a single dose) especially when the solution is concentrated, is capable of producing severe damage to the cauda equina or 'gumming' of the subarachnoid space." There is definite risk of introducing secondary infection when large numbers of injections are necessary. When there is brain abscess and resulting high intracranial pressure there is risk of producing a fatal pressure cone.

The usual treatment of meningitis is as follows: Intrathecally introduced penicillin should not be stronger than 2,000 units per cubic centimeter and should be given in doses of from 8,000 to 10,000 units. For the first two or three days injections should be given every twelve hours. Injection should be slow and the cerebrospinal fluid should be frequently aspirated to warm and dilute the mixture. If there is blocking of the cerebrospinal pathways (shown by slowing of flow of the cerebrospinal fluid and in difficulty of aspiration) injection should be made into the ventricle through burr holes. One case reported

23. Smith, H. V.; Schiller, F., and Cairns, H.: Discussion on Chemotherapy of Meningitis Secondary to Infection of the Ear and Nasal Sinuses, Proc. Roy. Soc. Med. **39**:613-628 (Aug.) 1946; J. Laryng. & Otol. **61**:313-337 (June) 1946.

was that of a man of 52 who had seven relapses of meningitis in thirteen weeks, but who recovered completely.

The usual treatment of brain abscess given was as follows: The abscess should be aspirated and penicillin injected into the abscess cavity. A more concentrated solution that may be used consists of 10,000 units per cubic centimeter; the quantity injected may be from 1 to 3 cc. Subdural abscess is dealt with by insertion of fine rubber catheters through several burr holes. These catheters are left open and pus aspirated and penicillin injected.

Macbeth said in the discussion that the operation on the mastoid in meningitis is no longer urgent. "Wait for a few days and in most of the acute cases operation is not needed."

Siirala [24] describes in great detail 4 cases of meningitis. In 2 cases infection entered by way of the labyrinth, and in 2 cases the purulent meningitis arose probably simultaneously by way of the labyrinth and by other pathways. Treatment consisted of administration of sulfonamide drugs and mastoidectomy, and not radical operation on the labyrinth. All 4 patients lived.

On the basis of his earlier experiments with guinea pigs to determine the effect of the sulfonamide drugs on purulent otitis interna, the author is of the opinion that cases of labyrinthogenic meningitis may be observed longer before resorting to trephinement of the internal ear. In none of the 4 cases was trephinement of the inner ear done. The hearing was completely destroyed in the affected ear in all cases.

Youngs and Lindsay [25] report the interesting case of a girl of 14 in whom signs of petrositis and labyrinthitis developed after she had had a discharging ear for two weeks. There was pain around the eye, dizziness, nausea and vomiting. Nystagmus was present. A simple mastoidectomy was done, and the nystagmus and other symptoms cleared up for three days. But at that time the nystagmus returned, and facial paralysis and spiking temperature suggesting lateral sinus thrombosis developed. The internal jugular vein was tied. The lateral sinus was found not to contain an obliterating thrombus. High levels of sulfadiazine were maintained, penicillin not being available in 1942. Recovery seemed complete, and the patient was in apparent good health for a little over a year. At that time symptoms of nausea, vomiting, vertigo, diplopia and a painful discharging ear developed. Search for a cell tract or fistula was not successful, and the patient died with symptoms of meningitis. Hemolytic streptococci were present at both attacks. Autopsy showed a tract of cells connecting the epitympanum

24. Siirala, U.: On the Treatment of Purulent Otitis Interna with Sulfonamides, Acta oto-laryng. **34**:200-216 (March) 1946.

25. Youngs, N. A., and Lindsay, J. R.: Suppurative Labyrinthitis, Ann. Otol., Rhin. & Laryng. **55**:43-54 (March) 1946.

and the necrotic area in the petrous pyramid. The illustrations of the pathologic process are excellent.

Lindahl [26] reported a case of a soldier 33 years of age who complained of pain in the back of the neck for three and a half months. He had had a mild otitis media following a cold, with spontaneous discharge of ten days' duration. The hearing remained slightly dull. Chemotherapy was not given. The drum appeared slightly thick but intact. His temperature was 101 F. Roentgenograms showed opacity of the mastoid cells with necrosis. Myringotomy was done and a little thick pus obtained. Mastoidectomy was done and all cells were found to be infected. An extradural abscess 2 inches (5.08 cm.) above and 1 inch (2.5 cm.) lateral to the external occipital process was found, and about 3½ ounces (104 cc.) of thick yellow pus was removed. Only Micrococcus catarrhalis grew on culture. The patient recovered.

Otte [27] presents 8 cases of cerebellar abscess in which there was one recovery. Since cerebellar abscess is such a common complication of either labyrinthitis or thrombosis of the lateral sinus, he urges watchfulness for cerebellar symptoms in these cases. The symptoms to be watched for are: (1) symptoms of intracranial pressure: intense occipital headaches, slow pulse, papilledema and vomiting; (2) symptoms of cerebral infection: stupor, mental changes, rapid loss of weight; (3) cerebellar symptoms, uncoordinated movements, and (4) septic symptoms of high fever, chills, etc. He advises approach to the cerebellar abscess by the mastoid route.

LABYRINTHITIS

Otte [28] also presented 2 cases of labyrinthitis. In 1 case in which granulation tissue was found over an erosion of the semicircular canal, the granulation was removed with no bad results. In the other case the entire labyrinth was removed and no complications developed. He states that operation on the labyrinth is not so important as in the past, as chemotherapy is so successful in all infections of the mastoid and complications.

In an article already quoted, Siirala [24] reported 4 cases of labyrinthitis: 3 occurred as complications of acute otitis media, and the fourth occurred during a flare-up of chronic otitis media brought on by swimming. All 4 cases cleared up with chemotherapy and operation on the mastoids; the labyrinth was not opened in any case. In 3 cases simple mastoidectomy was done and in the chronic case radical

26. Lindahl, J. W. S.: Large Extradural Abscess in the Occipital Region Secondary to Mastoiditis, J. Laryng. & Otol. **61**:245-247 (April) 1936.

27. Otte, G.: Cerebellar Abscess, Rev. otorrinolaring. **5**:192-215 (Dec.) 1945.

28. Otte, G.: Otitic Labyrinthitis, Rev. otorrinolaryng. **5**:216-235 (Dec.) 1945.

mastoidectomy was done. All 4 patients were completely deaf in the affected ear on recovery.

LATERAL SINUS THROMBOSIS

Reid and McGuckin [29] report an unusual case of cavernous sinus thrombosis occurring following mastoiditis and lateral sinus thrombosis. The infection spread by way of the superior petrosal sinus from the lateral sinus to the cavernous sinus. The patient was a 3 year old girl who was almost moribund on admission. Simple mastoidectomy was done; the lateral sinus was opened and packed off. The superior petrosal sinus was located and drained and large doses of sulfathiazole given. The patient recovered in two months. Penicillin was not available at that time. His other 3 cases of cavernous sinus thrombosis developed from furunculosis. The patients also recovered, 1 receiving sulfonamide compounds only, and the other 2 a combination of sulfonamide drugs and penicillin.

In an article already referred to, Seppälä [1] reports that in 678 surgical cases there were 35 cases of lateral sinus thrombosis. They all developed in mastoids with little pneumatic development.

PETROSITIS

Thornell and Williams [30] report in detail 3 cases of petrositis, in 2 of which meningitis developed. They advise that in operating for petrositis, drainage can usually be accomplished by following preformed cellular tracts to the involved region. As previously described by Williams, when a cellular pyramid is present, increased room around the labyrinth is usually present. If closed, apicitis is present and preformed tracts cannot be found by careful search, complete apicectomy by the Ramadier-Lempart technic is done at once.

If meningitis is present and no adequate explanation for its presence is found during the course of complete mastoidectomy, the dura should be uncovered both over the temporosphenoidal lobe and over the sinus and the cerebellum. If still no cause for symptoms is found, apicectomy should be done. They agree with Eagleton that chronic sphenoiditis should be suspected in apicitis.

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29. Reid, J. L., and McGuckin, F.: Cavernous Sinus Thrombophlebitis: A Report on Six Consecutive Recoveries, J. Laryng. & Otol. **61**:273-281 (May) 1946.

30. Thornell, W. C., and Williams, H. L.: Apical Petrositis: Medical and Surgical Management in Cases With and Without Complicating Meningitis, Arch. Otolaryng. **43**:393-401 (April) 1946.

Thornval [31] reports 3 cases of petrositis:

CASE 1.—A young man with otitis media of sixteen days' duration was operated on for mastoiditis. Pneumococcus type III was found on culture. He was discharged on the thirteenth day apparently cured. He returned three weeks later with meningitis and died the following day. Autopsy showed destruction of the superior part of the apex.

CASE 2.—A 28 year old man had acute otitis media for two months. Simple mastoidectomy was done. Two weeks later discharge became profuse, and pain started in the forehead and teeth. Paralysis of the abducens nerve developed. Radical mastoidectomy was done, and a small cavity was found between the cochlea and the eustachian tube. Symptoms of meningitis developed. A third operation was then done following Ramadier's technic. Pus was not found. The patient died a few hours later. Autopsy revealed that the pus was not quite reached.

CASE 3.—An 8 year old boy had had otitis media for four weeks. The roentgenogram showed a large defect in the apex. Simple mastoidectomy was done, but there was no improvement in pain. Vomiting developed. A radical operation was then done and the dura lifted up in the xygomatic region over an area 2 by 3 cm. Discolored yellow bone was seen, the chisel used and much pus obtained. A drain was inserted. No tracts were found before this approach. Recovery followed.

Knauer [32] reviewed the literature on Gradenigo's syndrome and found 399 cases reported to date. The syndrome has three outstanding symptoms: "Suppuration of the middle ear, intense pain and loss of function of the external rectus muscle." The acute purulent otitis extends by one of several routes to the tip of the petrous bone causing localized meningitis at that point. Paralysis of the external rectus muscle usually develops twenty to forty days after onset of the otitis media. The mortality was 18.6 per cent.

Knauer's patient was a boy of 10 in whom acute otitis media developed complicated by an acute frontal sinusitis that required external drainage. Prolonged administration of sulfathiazole brought about recovery without mastoidectomy. One hundred and ten references are given. He also mentioned that the pneumatic type of mastoid is more apt to be associated with this symptom complex than the sclerosed type.

PARALYSIS OF THE FACIAL NERVE

Kettel [33] reports 264 cases of paralysis of the facial nerve seen by himself during a period of thirty years at the Municipal Hospital in Copenhagen. The author states that when a positive response is seen

31. Thornval, A.: On the Treatment of Apicitis Suppurative, Acta oto-laryng. **34**:546-553 (Nov.) 1946.

32. Knauer, W. J.: Gradenigo's Syndrome: Review of Literature and Report of a Case Complicated by Frontal Sinusitis, Arch. Otolaryng. **44**:303-318 (Sept.) 1946.

33. Kettel, K.: The Course of Otitic Facial Palsy Under Adequate Treatment of the Disease of the Ear, Acta oto-laryng. **34**:311-316 (July) 1946.

in faradic testing the prognosis is good, but that a negative response is not necessarily an indication for immediate decompression.

After long observation of many cases, he draws the following conclusions:

1. The preoperative palsy in association with acute otitis media only requires adequate treatment of the ear disease. . . . Where in spite of a perfect mastoidectomy there is no sign of returning mobility after up to two months' observation, decompression should be done.

2. The preoperative palsy in association with chronic otitis media always indicates a radical mastoidectomy. If during operation a fistula is discovered on the facial canal, decompression is done simultaneously, otherwise the patient is observed up to two months before decompression is considered.

3. The postoperative palsy in direct conjunction with the operation always indicates immediate decompression and repair of the nerve at the site of the injury.

4. Postoperative palsy occurring after a free interval has a good prognosis.

Ogilvie [34] reported in detail 4 cases of facial paralysis developing as a result of acute otitis media. He states that he had more frequently seen facial paralysis develop as a complication of chronic otitis. In case 1, that of a girl of 5½ years, paralysis occurred on the third day and operation was done on the fourth day. In the second patient, a boy of 19 months, paralysis developed on the third day of infection and operation was done early. The third patient was a boy of 9, whose paralysis developed at the end of one week. The fourth patient was a man of 36 in whom paralysis developed after a flare-up one month after he had apparently been cured by a sulfonamide drug.

Recovery was complete in 3 cases and partial in the fourth case (which could not be followed). The author did not use faradism or splints in treatment, as he questions their value.

[Operation was done rather early in the first 3 cases. According to Kettle, Tickle, Decker and others, paralysis of the facial nerve appearing in the first week or so of an otherwise uncomplicated otitis media is not an indication for an early mastoidectomy.]

SECRETORY OTITIS MEDIA

Eagle [35] reports a group of 172 patients, all except 1 seen during the last four years. The patients complain of gradual onset of painless deafness and tinnitus. They usually state that they can feel a shifting as of fluid in the ear and that when the head is moved a certain way the deafness disappears. Eagle advises incision of the drum without

34. Ogilvie, K. R.: Facial Palsy Accompanying Acute Mastoiditis, with Comments on Chemotherapy in Acute Suppurative Otitis Media, Brit. M. J. **2**:263 (Aug. 24) 1946.

35. Eagle, W. W.: Secretory Otitis Media, Ann. Otol., Rhin. & Laryng. **55**: 55-67 (March) 1946.

anesthetic (which he says is practically painless) and aspiration of the fluid with a fine suction tip inserted through the incision.

Eagle suggests that a new virus disease has appeared, as he is "reluctant to admit that the disease had been prevalent in the years prior to 1941, and that our eyes are now seeing pathology which they had previously failed to see."

He saw only 1 case in which infection subsequently occurred; in most cases healing took place in a few hours. In 75 per cent of the cases there was recurrence, and of the other 25 per cent, only 3 required more than three additional myringotomies on the same ear.

TECHNIC

Boyden [36] performed forty-three simple mastoidectomies by way of the endaural approach. The first twenty were examined with the roentgen ray postoperatively, as well as preoperatively, to make sure all the mastoid cells were removed. No remaining cells were found. He favors the endaural route because: 1. There is less postoperative pain and easier convalescence. 2. The head dressing can be discarded much sooner. 3. The stay in the hospital is shortened. 4. Postoperative scars and occasional depressed scars are avoided. 5. All pathologic conditions of the temporal bone can be easily reached.

Simpson [37] gives the usual indications for the modified radical mastoid operation: (1) chronic otitis media with a pathologic process that can be eradicated through this approach, and (2) hearing that is worth saving in the affected ear.

His technic is nicely shown in two pages of excellent colored plates. There is no report of the number of ears treated by his technic. The postaural approach is used.

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36. Boyden, G. L.: Endaural Approach to the Mastoid for Complete Mastoidectomy, with Report of Forty-Three Operations, Ann. Otol., Rhin. & Laryng. **55**:543-548 (Sept.) 1946.

37. Simpson, W. L.: Modified Radical Mastoidectomy, Indications and Technique (Semiradical Mastoidectomy, Modified Tympanomastoidectomy, Heath Mastoidectomy, Bony Mastoidectomy), Arch. Otolaryng. **44**:157-159 (Aug.) 1946.

Correspondence

ACETYLSALICYLIC ACID AND BLEEDING FROM THE TONSILLAR WOUND

To the Editor:—The article by Livingston and Neary in the January 1948 issue of the ARCHIVES OF OTOLARYNGOLOGY entitled "The Question of Prothrobinopenic Hemorrhage from Post-Tonsillectomy Use of Chewing Gum Containing Acetylsalicylic Acid" prompts the following comments:

On the basis of a study of 45 children undergoing tonsillectomy, 29 of whom received chewing gum tablets containing acetylsalicylic acid for several postoperative days, and 16 of whom were given such tablets containing menadione in addition to the analgesic, these investigators concluded that "there is no indication that the routine post-tonsillectomy use of chewing gum containing acetylsalicylic acid may give rise to prothrombinopenic hemorrhage or that such use of this gum has any adverse effect on blood prothrombin."

Livingston and Neary make reference to my finding that salicylate-induced hypoprothrombinemia is an important factor in the causation of late tonsillar bleeding (Neivert, H.: Late Secondary Tonsillar Hemorrhage: I. Studies of Prothrombin and Vitamin K. ARCH. OTOLARYNG. **42:** 14 [July] 1945). They do not really deny this concept; yet by implication they assert the contrary. In fact, these investigators could not very well refute my claim since they gave the acetylsalicylic acid in the form of chewing gum, and I reported studies concerning the internal use of acetylsalicylic acid. However, I observed the prothrombinopenic effect of this analgesic when it was ingested and when it was taken in the form of chewing gum. Trials carried out for several persons, which have not yet been published, afforded evidence that a total daily internal dose of 27½ grains (1.78 Gm.) of acetylsalicylic acid increased the premedication prothrombin time values to the same degree as a total dose of 8 chewing gum tablets representing 28 grains (1.84 Gm.) of acetylsalicylic acid. (It is realized that the dose of the prothrombinopenic drug I used was somewhat larger than that administered by Livingston and Neary, but they worked on children and I on adults).

What strikes one first, when reviewing the Livingston-Neary report, is the exceedingly small number of patients from which these workers drew their conclusions. When I first published my experiences, I presented 283 patients who had received internally acetylsalicylic acid in combination with "synkayvite," a water-soluble vitamin-K-like substance (tetrasodium 2-methyl-1, 4 naphthohydroquinone diphosphoric

acid [ester]), compared with 16 patients given the combined medication studied by Livingston and Neary. Since then I have increased the number of tonsillectomized patients receiving acetylsalicylic acid in combination with "synkayvite" to 622, with results as gratifying as were those in the group reported originally. Preston (*Virginia M. Monthly* **73**: 252, 1946), investigating the etiologic relation between salicylate medication and incidence of late tonsillar hemorrhage, realized also that only a study of large numbers of cases will throw light on the problem under investigation. As compared with 45 patients presented by Livingston and Neary, he observed 1,808 patients and confirmed my findings.

The series presented by Livingston and Neary is too small to warrant expression of the results on a percentage basis. However, had the groups of patients studied by them been ten times as large, and had results been analogous, the incidence of hemorrhage in the patients receiving chewing gum containing acetylsalicylic acid, on the one hand, and in those receiving the gum and menadione on the other, would have been between 6 and 7 per cent.

If such hypothetic findings are correlated with the findings recently reported by Fox and West (*Laryngoscope* **57**: 564, 1947), it appears that the results of both these independently working groups of investigators are in agreement: They both encountered a fairly high incidence of late tonsillar hemorrhage irrespective of whether or not they added vitamin K to the chewing gum containing acetylsalicylic acid. As had been pointed out previously (Neivert, H.: Letter to the Editor, *Laryngoscope* **57**: 725, 1947), one may conclude from the data presented by Fox and West that chewing gum containing acetylsalicylic acid predisposed to bleeding caused by some mechanism other than salicylate-induced hypoprothrombinemia and therefore one which cannot be prevented by vitamin K. The results of Livingston and Neary, if corroborated in appreciably larger numbers of patients, would tend to augment my suspicion that acetylsalicylic acid has a local untoward effect on the tonsillar wound.

March 12, 1948

HARRY NEIVERT, M.D., NEW YORK.

Obituaries

GEORGE E. SHAMBAUGH, M.D.

1869-1947

IN THE DEATH of Dr. George E. Shambaugh Sr., on Nov. 30, 1947, the otologic profession in America lost one of its most distinguished members. Although Dr. Shambaugh had been retired for a number of years on account of physical disability, he had been, almost since graduation, one of the eminent pioneers in otologic research, an evaluation of which, by his eminent colleague, Dr. J. G. Wilson, follows.

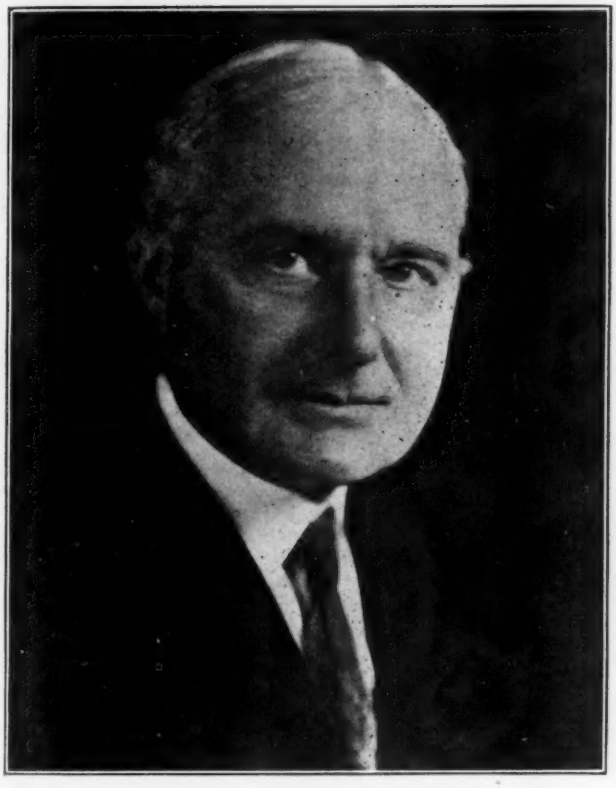
Almost from the beginning Dr. Shambaugh devoted himself to a careful research study of the physiology and anatomy of the internal ear, and in addition had an international reputation as a teacher and clinician of the first water.

He was the founder and first editor-in-chief of the ARCHIVES OF OTOLARYNGOLOGY, and organizer of the American Board of Otolaryngology. He was a fellow of all the senior societies concerned with his specialty, and had been president of both the American Otological Society and the American Laryngological Association.

For many years he was professor of otology in the Rush Medical College and in the University of Chicago, and early in his career had been secretary and then chairman of the Section on Otolaryngology of the American Medical Association.

His research work laid the foundation for, and pointed the way for the many eminent members of the profession who have followed in his footsteps.

GEORGE M. COATES, M.D.



GEORGE E. SHAMBAUGH, M.D.

1869-1947

Special Reports

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION

As of July 1, 1948

Aurex (Semi-Portable)	J. A. M. A. **109:** 585 (Aug. 21) 1937.
Aurex Model C-B and Model C-A	J. A. M. A. **120:** 535 (Oct. 17) 1942.
Aurex Model F	Report not yet published.
Aurex Model H Mfr. Aurex Corporation 1117 North Franklin Street Chicago	J. A. M. A. **136:** 1099 (April 24) 1948.
Beltone Mono-Pac	J. A .M. A. **130:** 637 (March 9) 1946.
Beltone Harmony Mono-Pac Mfr. Beltone Hearing Aid Company 1450 West Nineteenth Street Chicago	J. A. M. A. **133:** 543 (Feb. 22) 1947.
Dysonic Model 1 Mfr. Dynamic Hearing Aids 43 Exchange Place New York 5	Report not yet published
Electroear Model C Mfr. American Earphone Company, Incorporated 10 East Forty-Third Street New York 17	J. A. M. A. **136:** 769 (March 13) 1948.
Maico Type K	J. A. M. A. **129:** 32 (Sept. 1) 1945.
Maico Atomeer Mfr. Maico Company, Inc. North Third Street Minneapolis	J. A. M. A. **133:** 542 (Feb. 22) 1947.
1947-Mears Aurophone Model 98 Mfr. Mears Radio Hearing Device Corporation 1 West Thirty-Fourth Street New York	Report not yet published.

Microtone T-4 Audiomatic Mfr. Microtone Company 4602 Nicollet Avenue Minneapolis 9	J. A. M. A. **136:** 109 (Jan. 10) 1948.
Otarion, Model A-1	J. A. M. A. **115:** 1101 (Sept. 28) 1940.
Otarion, Model A-3	J. A. M. A. **132:** 1071 (Dec. 28) 1946.
Otarion, Model A-4 J & S	J. A. M. A. **132:** 925 (Dec. 14) 1946.
Otarion, Model E-1 Mfr. Otarion Hearing Aids 159 North Dearborn Street Chicago	J. A. M. A. **136:** 108 (Jan. 10) 1948.
Paravox Models VH and VL	J. A. M. A. **132:** 79 (Sept. 14) 1946.
Paravox Model XT	J. A. M. A. **134:** 365 (May 24) 1947.
Paravox Model XTS Mfr. Paravox, Inc. 2056 East Fourth Street Cleveland 15	J. A. M. A. **136:** 109 (Jan. 10) 1948.
Radioear Masterpiece (carbon type)	J. A. M. A. **117:** 2169 (Dec. 20) 1941.
Radioear Model 45-CM	J. A. M. A. **126:** 1151 (Dec. 30) 1944.
Radioear Model 45-M-magnetic air conduction receiver	J. A. M. A. **127:** 219 (Jan. 27) 1945.
Radioear Model 45-M-magnetic bone conduction receiver Mfr. E. A. Myers & Sons 306 Beverly Road Mount Lebanon Pittsburgh	J. A. M. A. **127:**27 (Jan. 6) 1945.
Ravox (Semi-Portable) Mfr. Zenith Radio Corporation 6001 West Dickens Avenue Chicago	J. A. M. A. **113:** 18 (Oct. 28) 1939.
Silver Micronic Hearing Aid Mfr. Micronic Corporation 101 Tremont Street Boston 8	J. A. M. A. **135:** 159 (Sept. 20) 1947.
Solopak Hearing Aids Mfr. Allen-Howe Electronics Corporation 150 Main Street Peabody, Mass.	J. A. M. A. **136:** 768 (March 13) 1948.
Sonotone Audicles Nos. 530, 531 and 533	J. A. M. A. **123:** 837 (Nov. 27) 1943.

Sonotone Model 600	J. A. M. A. **131:** 523 (June 8) 1946.
Sonotone Model 700	J. A. M. A. **135:** 838 (Nov. 29) 1947.
Mfr. Sonotone Corporation Elmsford, N. Y.	
Telex Model 22	J. A. M. A. **134:** 605 (June 14) 1947.
Telex Model 612	J. A. M. A. **114:**1634 (April 27) 1940.
Telex Model 900	J. A. M. A. **117:** 1978 (Dec. 6) 1941.
Telex Model 1020	J. A. M. A. **117:** 2072 (Dec. 13) 1941.
Telex Model 1550	J. A. M. A. **126:** 705 (Nov. 11) 1944.
Mfr. Telex, Inc. Minneapolis 1	
Trimm Vacuum Tube Model 300	J. A. M. A. **133:** 542 (Feb. 22) 1947.
Mfr. Trimm Inc. 400 West Lake Street Libertyville, Ill.	
Unex Model "A"	J. A. M. A. **134:** 254 (May 17) 1947.
Mfr. Nichols & Clark Hathorne, Mass.	
Vactuphone Model 3	J. A. M. A. **136:** 769 (March 13) 1948.
Mfr. Allen-Howe Electronics Corporation 150 Main Street Peabody, Mass.	
Western Electric Ortho-tronic Model	J. A. M. A. **121:** 1283 (April 17) 1943.
Western Electric Model 63	J. A. M. A. **131:** 895 (July 13) 1946.
Western Electric Model 64	J. A. M. A. **134:** 605 (June 14) 1947.
Western Electric Models 65 & 66	J. A. M. A. **137:** 534 (June 5) 1948.
Mfr. Western Electric Co. Inc. 120 Broadway New York 5	
Zenith Radionic Model A-2-A	J. A. M. A. **127:** 159 (Jan. 20) 1945.
Zenith Radionic Model A-3-A	J. A. M. A. **127:** 159 (Jan. 20) 1945.
Zenith Radionic Model B-3-A	J. A. M. A. **127:** 158 (Jan. 20) 1945.
Zenith Model 75	J. A. M. A. **135:** 773 (Nov. 22) 1947.
Mfr. Zenith Radio Corporation 6001 West Dickens Avenue Chicago	

All the hearing devices listed in this report except the one designated as of the carbon type employ vacuum tubes.

Abstracts from Current Literature

Ear

A Hearing Aid for the Relief of Tinnitus Aurium. Maurice Saltzman and Matthew S. Ersner, Laryngoscope **57**:358 (May) 1947.

Tinnitus aurium is frequently difficult or impossible to relieve. If it is associated with conductive deafness, the person may be hearing noises created within his own body. It has been found that the fenestration operation often relieves tinnitus. The reason for this is that the operation allows outside sound to enter in, masking the tinnitus. The hearing aid, adjusted properly, lets in outside sound which likewise eliminates the tinnitus or at least makes it bearable.

Case reports are included.

Hitschler, Philadelphia.

The Fenestration Operation: Observations on the Evaluation of Hearing Tests. J. R. Lindsay, Laryngoscope **57**:367 (June) 1947.

Observations are made on the establishment of criteria that may be used in the evaluation of the possibilities for improvement in each case, or, in other words, the selection of cases in which the hearing apparatus will respond satisfactorily after the fenestration operation, and on the maintenance of the fenestra.

Most of the cases in which the fenestra closed were among the first Lindsay performed. The probability is that in these cases closure was due to faulty technic. Sources of error in technic are mentioned.

In a fairly large group of cases there was a decrease of hearing in the low tones in the first six months postoperatively. Such a decrease may represent a partial closure of the fenestra. These cases can be considered successes for the most part because of the maintenance of the hearing gain at 2048 and 4096.

Various types of hearing losses are shown and compared with postoperative losses. For a good postoperative result there should be good bone conduction in the speech frequency area preoperatively. The bone conduction values at 1024 and 2048 give the best indication of the success or the failure of the operation.

Hitschler, Philadelphia.

Allergic Deafness. Abner M. Fuchs and Ralph Almour, New York State J. Med. **47**:1397 (June 15) 1947.

The authors estimate that 10 per cent of all people suffer from major allergies and that some 50 per cent have "minor allergic reactions." They note the prevalent trend to classify an enormous number of conditions under this heading. A man who suffered from hay fever, bronchial asthma, migraine and vertigo went into shock on one occasion after self administration of a pollen extract and was "unconscious" for two hours (?). Following incision of the right drum in childhood, he had been deaf in that ear. Later he lost hearing in the left ear, but it returned within a few hours. Recently these on and off attacks of loss of hearing recurred once a month, then every four days. He could foretell the onset. There was "itching inside the ear." He spoke of a gushing of fluid into the throat, after which he heard better. Sensitization to grasses, weeds, orris root, dust, feathers, animal excrescences (danders), etc., was "marked." There were also reactions to cereals and other foods. Fish, tomato and ketchup when eaten to excess "produced a marked deafness which lasted three days." Subcutaneous

injection of "slow epinephrine hydrochloride" relieved the deafness within a half hour. The authors infer that there is an expression of angioneurotic edema in the right ear which has permanently destroyed the auditory nerve. The left is still "reversible" through treatment with epinephrine, but permanent loss may be expected here, too. An effort is being made to desensitize this patient, but no opinion is expressed as to the efficacy.

VOORHEES, New York.

ACUTE NONSUPPURATIVE ENCEPHALITIS OF AURAL OR NASAL ORIGIN. JEAN PIQUET, Acta oto-laryng. **35**:211 (May-June) 1947.

Under the title of "acute nonsuppurative encephalitis," Piquet considers a number of conditions dissimilar as to their clinical course and their anatomic manifestations. Among these are the peripheral encephalitis accompanying acute meningitis, the meningoencephalitis of infected cranial wounds, the epidemic encephalitis and the encephalitis of infectious diseases. Then there is the encephalitis which precedes a cerebral abscess. Finally, there is the acute nonsuppurative encephalitis, called hemorrhagic, which originates from the ear. This condition has been studied by Jacks, Jansen, Oppenheim, Borries and others.

As the condition called acute hemorrhagic encephalitis usually ends in recovery, little is known about its anatomopathologic character. It is set up by acute otitis or an exacerbation of chronic otitis. It may accompany premastoiditis lesions, and it may result from the complications of mastoiditis, such as extradural abscess and thrombosis of the lateral sinus. It has never been ascribed to sinusitis, but Piquet reports a case in which it was due to chronic frontal sinusitis.

Clinically, the encephalitis manifests itself by the signs of a cerebral abscess without abscess formation. The onset is more or less rapid, at times even apoplectiform. The elevation of temperature varies, and it may be entirely absent. Focal signs frequently constitute the first evidence of the disease. These consist of hemiplegia, usually a contralateral monoplegia, aphasia, facial paralysis of the opposite side or at times of the same side, and motor paralysis of the eye. Cerebellar symptoms are rarely seen. Convulsive seizures are rare. Headache is generally slight. Slow cerebration is frequent, but the deep coma of cerebral abscess is usually absent. Signs of meningitis are frequent but variable. The cerebrospinal fluid varies considerably. It may present the characteristics of purulent aseptic meningitis or those of serous meningitis. It may be hemorrhagic or clear and under more or less pressure. Occasionally but rarely the cerebrospinal fluid may be normal.

The exact nature of the benign forms of hemorrhagic encephalitis from which patients recover is not known. With respect to the more severe forms to which patients succumb, Piquet calls attention to one of his previous communications (1937) in which he distinguished four distinct varieties:

1. Encephalitis with diffuse necrosis of a part of the brain, associated with edema of the cerebral substance. The cerebral vessels are thrombosed, but one does not find any polymorphonuclear infiltration. Death is inevitable.

2. Diffuse encephalitis with multiple foci. The infection is less severe and is characterized by formation of small miliary abscesses without great tendency to coalesce. This condition also ends fatally.

3. Encephalitis with limited necrosis of the cerebral mass, without suppuration. The cerebral tissue is transformed into a reddish or reddish yellow soft pulpy mass which runs out of the trocar as a semiliquid substance. This material is bloody, and bloody effusions are found in the subarachnoid spaces and also in the perivascular spaces. The patients frequently recover.

4. The edematous and generally benign encephalitis. This serous infiltration can take place with extreme rapidity and bring on death within an hour by cerebral compression. This variety has been reported as "serous meningo-encephalitis" by Korrner.

The first two varieties are very serious, whereas the last two are usually benign. The last two are indistinguishable clinically and can be differentiated only by aspiration. However, the tissue involved by the serous type is often so hard that fluid cannot be aspirated. The transitory paralyses caused by this type are explained by the absorption of the edematous fluid.

Piquet reports 3 cases of hemorrhagic encephalitis: The first patient was a child of 4 years. Early in the course of acute otitis, encephalitis appeared. Recovery was complete. The second was a woman aged 58. During the course of acute otitis media, encephalitis appeared. A complete recovery was made. The third patient was a man aged 37 with acute frontal sinusitis. The frontal sinus was trephined. About three weeks after the onset of the sinusitis, symptoms of encephalitis appeared. The man made an uneventful recovery. GROVE, Milwaukee.

OTOGENIC BRAIN ABSCESS. L. B. W. JONGKEES, Acta oto-laryng. **35**:225 (May-June) 1947.

Sulfonamide drugs and penicillin have not particularly altered the course of brain abscess when administered orally or intramuscularly.

The diagnosis of brain abscess may be easy if signs of increased intracranial pressure (headache, papilledema, slow pulse, paresis of the sixth nerve or specific alterations of the cerebrospinal fluid) and local signs pointing to the cerebellum or the temporal lobe are present. On the other hand, a very large abscess can be present without any symptoms or localizing signs.

The composition of the cerebrospinal fluid seems to provide the best help for making a diagnosis but some otologists fear to use a lumbar puncture in arriving at the diagnosis for fear that the cerebellum may be crowded into the foramen magnum. Jongkees feels that the removal of a small amount of fluid is justified if the diagnosis is difficult.

The treatment of an otogenic abscess of the brain is surgical. Neurosurgeons prefer the extra-aural route, but otologists prefer the aural route because thereby the route of the infection may be traced. Some surgeons advise surgical intervention as soon as the diagnosis is made, while others prefer to wait four or five weeks until the abscess has been encapsulated. The danger of this procedure is that the patient may die while the capsule is forming. The type of surgical procedure varies a good deal, but the general principle followed is to extirpate the abscess within its capsule or to drain it.

Jongkees reports 2 cases of brain abscess, one of abscess of the cerebellum and one of abscess of the temporal lobe. In the latter case recovery occurred after a conservative form of treatment, which consisted in puncturing the abscess and allowing the pus to escape through the needle and then introducing penicillin into the abscess through the puncture needle. In each of these cases the abscess complicated chronic otitis. GROVE, Milwaukee.

ALLERGIC INVESTIGATIONS OF CHRONIC OTITIS. HJALMAR KOCH, Acta oto-laryng., 1947, supp. 62.

This supplement is an exhaustive study of 222 patients with 262 ears presenting chronic otitis media. Fifty-two ears presented an eosinophilic type of secretion; the remainder, a noneosinophilic type. The macroscopic appearance of the two

types of secretion was markedly different. The noneosinophilic secretion observed in cases with a central perforation of the tympanic membrane was initially purulent or mucopurulent. As treatment progressed, the secretion became mucoid, but never very fibrinous, and was easily removed by rinsing and drying. Marginal perforations were chiefly characterized by a purulent secretion which persisted during treatment. This was especially true when cholesteatoma was present.

The eosinophilic secretion of chronic otitis was characteristic. It was clear or serum-like and so viscid that it had to be removed with forceps. This macroscopic appearance was so characteristic that the cytologic picture could be predicted in most of the cases.

The microscopic picture of the noneosinophilic secretion was dominated by neutrophils, interspersed with lymphocytes and monocytes. Eosinophils were scarce. During the treatment of patients with a central perforation of the tympanic membrane a shift in the lymphocytic direction took place. In those with the marginal perforations the cell picture remained unchanged.

In the eosinophilic secretion many eosinophils could be seen either localized in clumps in the mucus streaks or dispersed throughout the preparation. If eosinophils were not found on the first or the second examination of the eosinophilic type of secretion and appeared only after sulfonamide medication, the drug was suspected as the cause of the eosinophilia.

In the cases in which the secretion was noneosinophilic, the mucosa of the ear was slightly or only moderately swollen. In the cases in which the secretion was eosinophilic, the mucosa was always edematous and, as a rule, markedly so. In the eosinophilic stages the mucosa was pale rose in color with some cyanotic interspersions, as in vasomotor rhinitis.

Under the microscope the mucous membrane of the ear in the cases in which the secretion was noneosinophilic showed chronic inflammation with round cell infiltration. There was often marked fibrosis. In the polypi and granulations neutrophilic leukocytes were seen, but usually no eosinophils.

In the cases of chronic otitis in which the secretion was eosinophilic, a large amount of eosinophilia of pathologic tissue was found, and there was definite accord between the eosinophilia of the secretion and that of the tissue. Allergic rhinitis was common in patients with eosinophilic otitis. The time of healing was more prolonged in the cases of eosinophilic otitis than in those in which eosinophils were not shown.

In regard to treatment, 18 ears healed after antiallergic treatment, and the allergic rhinitis disappeared at the same time. In 6 cases in which elimination of the allergen had no effect, the ears healed after desensitization. In 3 cases the ears healed after combined elimination of allergens and desensitization. In these cases there seemed to be strong reasons for assuming that the exacerbation of the chronic otitis was due to an allergen.

No difference was noted in the structure of the air cell system between the cases of eosinophilic and the cases of noneosinophilic otitis. GROVE, Milwaukee.

Pharynx

FAUCIAL DIPHTHERIA IN ADULTS WITH REFERENCE TO THE EARLY DIAGNOSIS: REPORT OF A CASE WITH COMPLETE HEART BLOCK. JOSEPH GOLDSTEIN and PHILIP L. CALCAGNO, New York State J. Med. **47**:1511 (July 1) 1947.

The incidence of diphtheria seems to have increased in the United States during 1944-1945. "Many returning American troops are undoubtedly carriers of virulent diphtheria bacilli." They may be responsible for spreading the contagion even though they may not have had any symptoms of the disease. At the Willard

Parker Hospital the number of cases seen is higher now than during the past five years. Of especial importance is the fact that diphtheria occurring in adults may not even be suspected, much less recognized, until late. The instance of a 33 year old white man is cited in detail. He is not referred to as "a veteran," since he was "rejected" from the draft. "A mass of organized tissue appeared to project from the oral pharynx. It extended forward over the soft palate and included half of the hard palate. The portion adjacent to the oral pharynx seemed to be attached to the right tonsil. The surrounding tissues of the throat for only a short distance beyond the mass were a lurid red color. The anterior edge of the mass was lying free in the mouth just above the tongue."

Cultures of material taken from the nose and the throat on admission revealed only Staphylococcus aureus. Later cultures showed Corynebacterium diphtheriae of the mitus type, toxic for guinea pigs. The patient died on the tenth day.

The diagnoses suggested on inspection were: neoplasm, fungous infection, some blood dyscrasia. Smears and cultures "fooled" the investigators, since the diphtheria organism seems to have been deep in the adventitious membrane and not on the surface, which yielded merely the incidental Staph. aureus.

The authors set down five points which ought to help in correctly diagnosing such a massive diphtheritic infection: (1) There is a demarcated area between the "normal" mucous membrane and the exudate; (2) there is a seeming extension of the exudate at subsequent inspections; (3) the membrane extends beyond the faucial pillars; (4) there is involvement of the uvula; (5) the soft palate shows edema with only moderate surrounding redness; (6) pyogenic micro-organisms cause a more generalized and extended hyperemia.

Certain misconceptions need to be corrected: (1) the belief that pain is not common in diphtheritic infections (edema of soft parts usually gives pain here as elsewhere); (2) the idea that a low temperature shuts out diphtheria (adenocellulitis, mixed infection, may elevate the temperature); (3) dependence on a "musty odor" (necrosis is not essentially "musty"). Some diphtheritic infections closely simulate follicular tonsillitis, and the diphtheria bacillus may be found in the follicles, but coalescence and extension beyond the pillars with membrane formation should arouse suspicion of a generalized pharyngeal diphtheria.

Frequent cultures should be made of material taken not only from the surface but from below the membrane as well. The authors state that "only after cultures taken at forty-eight-hour intervals are returned with negative reports is the patient considered non-diphtheric." The point is made that diphtheria can occur in patients whose reactions to the Schick test are negative, but complications are then unusual. The question is asked by these authors why the Schick test is not done in the armed forces, especially at induction. This test is a "booster" to those patients who have previously been inoculated. The example is given of a 24 year old veteran who reacted positively to the test and who was admitted to the Willard Parker Hospital one week after being dismissed from the Army with faucial diphtheria. He died five weeks later from severe myocarditis! "In view of these facts it would seem advisable to have Schick tests done routinely on Army personnel."

VOORHEES, New York.

Larynx

THE USE OF TRACHEOTOMY IN BULBAR POLIOMYELITIS. DANIEL F. GLASER, J. Pediat. **27**:560 (Dec.) 1945.

Bulbar poliomyelitis is that form of the disease in which the infection involves the brain stem and gives rise to clinical impairment of any of the cranial nerves.

The prognosis becomes grave when the hypoglossal, pharyngeal and vagus nuclei are involved.

The causes of death from central paralysis include failure of the respiratory center and cardiovascular collapse. Because of the inability to swallow, the nasal and buccal secretions form a "puddle" in the posterior part of the pharynx. Aspiration pneumonia and atelectasis frequently result. Postural drainage and tracheotomy aid in removing and in by-passing this puddle.

Because of the accumulated secretions in the posterior part of the pharynx, aspiration pneumonia and atelectasis are almost certain to follow should the patient be placed in a respirator. However, if an oxygen supply is attached directly to the tracheotomy tube, a valve mechanism is set up. There is positive pressure in the oxygen supply line which makes exhaling through it impossible. The exhaled gases tend to escape around the tracheotomy tube and out through the nasopharynx. This tends to force the accumulated secretions out where they may be removed by suction. During inspiration the supply tends to be derived from the oxygen supply, and consequently the "puddle" of secretions is by-passed. In order that the oxygen supply might be connected directly to the tracheotomy tube, the tube was altered, a nipple being soldered to the inside tube.

Two patients with failure of the respiratory center due to bulbar poliomyelitis were treated with the valve. The first patient was removed from the respirator after eleven days. After six months there is still some degree of residual paralysis, but the patient is otherwise in good health. The second patient died on the eighth hospital day of cardiovascular collapse.

From August to December 1944, 8 patients with severe bulbar poliomyelitis were seen. Of these the only one to survive was the first patient mentioned, who was removed from the respirator after eleven days.

AUTHOR'S ABSTRACT. [AM. J. DIS. CHILD.]

CONTACT ULCER OF THE LARYNX: A CASE REPORT. HARRY C. ROSENBERGER, Laryngoscope **57**:272 (April) 1947.

Contact ulcer of the larynx has only recently (1928) been recognized. The ulcer is on the posterior end of one or both vocal cords. Movement of the cords is usually normal. The symptoms include hoarseness, tired voice, discomfort and cough.

The cause, the differential diagnosis, the pathologic aspects and the treatment are discussed. A case report is included.

HITSCHLER, Philadelphia.

ONE STAGE RESECTION OF TOTAL CERVICAL ESOPHAGUS, LARYNX, BASE OF TONGUE, HYPOPHARYNX, CERVICAL TRACHEA AND BILATERAL CERVICAL LYMPH NODE CHAINS FOR CARCINOMA PRIMARY IN THE CERVICAL ESOPHAGUS: RECONSTRUCTION OF CERVICAL ESOPHAGUS. ALEXANDER BRUNSCHWIG and EDWARD CAMP, Laryngoscope **57**:305 (May) 1947.

Brunschwig and Camp describe a one stage resection of the total cervical portion of the esophagus, the larynx, the base of the tongue, the hypopharynx, the cervical portion of the trachea and the bilateral cervical lymph node chains for carcinoma primary in the cervical portion of the esophagus. The esophagus was reconstructed by means of a cutaneous flap. The larynx had to be sacrificed because of the removal of the neoplasm. There was no difficulty in deglutition in spite of the fact that no special exercises were carried out by the patient.

HITSCHLER, Philadelphia.

A Case of Esophageal Foreign Body with Mediastinitis and Other Complications. Edward H. Lyman, Laryngoscope **57**:400 (June) 1947.

It is recognized that a foreign body in the esophagus is a dangerous condition. There have been many fatalities due to impaction, instrumentation for removal, or complications caused by the body piercing or eroding the esophageal wall. In the case herein reported, the foreign body pierced the esophageal wall with resulting mediastinitis, subcutaneous emphysema and other complications.

Several authors have reported cures obtained with the aid of massive doses of penicillin. Such doses were used in this case, together of course, with the removal of the foreign body and other therapeutic measures. Lyman feels that the determining factor in the patient's ultimate recovery was the use of massive doses of penicillin. The case is reported in detail.

Hitschler, Philadelphia.

The Classification of Carcinoma of the Larynx. T. E. Walsh, Laryngoscope **57**:414 (June) 1947.

Classification of disease should "improve our understanding of the therapeutic approach to each variety and the prognosis of such treatment." With this in mind, Walsh classifies cancer of the larynx as (1) intrinsic, (2) endolaryngeal, (3) subglottic and (4) extrinsic or extralaryngeal.

Walsh explains just exactly what he means by each of these divisions and why he feels that the treatment and the prognosis are better differentiated by such a classification.

Hitschler, Philadelphia.

Nose

The Histological Effect of Repeated Application of Certain Nose Drops to the Nasal Mucous Membrane of Rabbits. Robert E. Ryan, Ann. Otol., Rhin. & Laryng. **56**:46 (March) 1947.

The author used two preparations of vasoconstrictors, one containing a sulfonamide compound and the other without it. The work was done on rabbits. Histologic study showed that cilia were present after the first preparation had been applied for three days, but none were found after the fifth day. Dilatation of the blood vessels of the mucosa occurred as early as the third day and continued until the later stage of the study, when constriction occurred. Inflammatory cells were found in the early stages, and their number increased until the fourth week, after which they gradually disappeared. Epithelial cells were damaged as early as the fifth day. During the third week the epithelial cells on the surface showed degenerative changes which persisted through the tenth week. The edema in the epithelial layer of cells increased in direct proportion to the length of time the preparation was used. In the latter stages of the study these cells became filled with mucus and resembled goblet cells. Finally, the surface became entirely of the stratified squamous type. The subepithelial layer showed thickening and fibrosis in the third week; edema was present in the fourth week and remained throughout the study.

With the second preparation destruction of the cilia was noted after eight days and thereafter throughout the study. The blood vessels were dilated in the early stages; after forty days they became constricted and finally sclerosed. Inflammatory cells were present from the earliest stages but tended to disappear in the later stages and were not noted in numbers after the fiftieth day. The epithelium was

damaged during the first week, showed edema and appeared filled with mucus. In the fifth week the layer became disorganized and bore little resemblance to normal. About the seventeenth day the surface was of a stratified squamous type. In the later stages the subepithelial layer became thicker and denser than normal, and fibrosis was present.

(Preparation 1 was a 0.125 per cent solution of dl-desoxyephedrine hydrochloride in a stabilized aqueous vehicle, 1.25 per cent of anhydrous sulfathiazole sodium and 1.25 per cent of sulfadiazine sodium.)

(Preparation 2 was a solution of naphazoline hydrochloride ["privine hydrochloride"] in an isotonic aqueous solution with a p_H of 6.2. It is put up in a 0.1 per cent and a 0.05 per cent solution. The weaker one was used in this study.)

M. V. MILLER, Philadelphia.

A NEW TECHNIQUE FOR THE INSERTION OF A COLUMELLAR STRUT. MATTHEW S. ERSNER and MAURICE H. ALEXANDER, Eye, Ear, Nose & Throat Monthly **26**:257 (May) 1947.

In the insertion of a columellar strut it is the aim of the rhinoplastic surgeon to achieve primarily functional efficiency of the nose. However, it is highly desirable to achieve a maximum of cosmetic perfection and to combine precision with safety.

Until our newer technic was developed, the rhinoplastic surgeon had resort to three other methods of inserting a strut into the columella. These were (1) the Gillies method, (2) the "elephant trunk" or Gillies method and (3) the Fomon method. However, each had its disadvantages.

We now call the attention of rhinologic surgeons to our technic, the Ersner-Alexander method, which offers many advantages.

We create an almost completely closed bed between the columellar cartilages into which the batten or strut is inserted. Thus, there is less possibility of infection or of movement of the graft. We also contend that use of live cartilage taken from the patient's septum at the time of operation is a great advantage. In no instance has there been absorption, disintegration or extrusion of the strut. There has been no interference with blood and lymph supply, nor has there been infection or hematoma in the columella. Last but not least, there is minimal mechanical manipulation. Thus, it may truly be said that here is a technic by which to insert a cartilaginous strut in the columella that has the advantages of being performed under direct vision with no external scar, with speed and precision and with safety as well as affording a maximum of cosmetic perfection.

AUTHORS' ABSTRACT.

A PRECISE METHOD OF APPROXIMATING THE MESIAL CRURA. MATTHEW S. ERSNER, MAURICE H. ALEXANDER and IRVING A. RUSH, Eye, Ear, Nose & Throat Monthly **26**:263 (May) 1947.

The authors have devised a precise method of approximating the crura mediale nasi. In it the anterior tips of the crura are sutured together before they are freed and delivered. The sutures are placed intranasally while the columella is still in the undistorted midline position, assuring accuracy of placement. This technic is a great improvement in that it has definite advantages over the former technic of the one-sided delivery in which the surgeon was forced to gage empirically, with no assurance of accuracy, and in which there were tension and buckling when the sutured crural tips were returned to the midline.

The authors recommend this new and practical method as a simple solution of the problem:

A white silk suture thread is first dipped into, and impregnated with, a water-soluble solution of methylene blue. It is then inserted through the anterior tips of the crura and pulled on through and out and discarded. The minute perforations caused by the passage of the needle and the suture material are thus temporarily stained by the water-soluble dye. These temporary tattoo marks fix definite landmarks for trimming the crura, which may be gaged with complete accuracy, thus permitting the cartilage to be cut in relation to the visible stained marks. This method is recommended because this procedure eliminates the need for any empiric gaging on the part of the surgeon when there is assurance of accuracy and there is no tension when the sutured tips of the crura are returned to the midline.

AUTHORS' ABSTRACT.

SURGERY OF THE FRONTAL SINUS. HENRY M. GOODYEAR, Laryngoscope **57**:340 (May) 1947.

Goodyear favors trephining the frontal sinus in certain cases of acute infection. The opening is made with a nail trephine just posterior to the medial part of the orbital ridge. Sometimes in adults there are recurring attacks of acute frontal sinusitis with severe pain. It is often advisable to operate on these patients for the relief of the pain. The technic of the operation is given.

Surgical treatment of chronic frontal infections is described. Goodyear advocates the use of the Ingals self-retaining gold tube following the Jansen-Lynch operation.

Other operative procedures are mentioned, including an intranasal method for the removal of mucoceles of the ethmoid and frontal sinuses.

HITSCHLER, Philadelphia.

CELLULITIS OF THE CHEEK COMPLICATING NASAL FRACTURE. ERNEST J. ELSBACH, New York State J. Med. **47**:1267 (June 1) 1947.

A 20 year old white woman crashed against the "dashboard" of an automobile when the brakes were applied by the driver, with resultant nasal fracture, abrasions, depression of the bridge, crepitus, and leftward deviation of the external nose. About forty-eight hours later, there was a temperature of 105 F., and pus was draining from the right nostril. Whether infection had entered from a sinus or through the external wound was not determined. The roentgen pictures showed little, but cellulitis of the right cheek was evident, and hemolytic streptococci were isolated from the pus. An initial dose of 40,000 units of penicillin was given intramuscularly, followed by 25,000 units every three hours. In addition, 1 Gm. of sulfadiazine was administered with sodium bicarbonate every four hours on the theory that it penetrates into the subarachnoid space better than penicillin, thus helping to prevent meningitis. The patient was discharged from the hospital ten days after admission "cured," but in need of plastic correction when sufficient time has elapsed.

Nasal fracture without skull fracture is usually of minor importance, says the author, although secondary meningitis has been reported. No reference to cellulitis of the cheek and abscess of one nostril has been found in the literature of the past ten years. Elsbach says that before the advent of penicillin and sulfonamide compounds, extensive surgical correction would have been necessary, and he gives full credit to chemotherapy without surgical intervention in this instance.

VOORHEES, New York.

A Simplified Technic of Treating Sinusitis with Penicillin Aerosol, with a Description of a Foot Pump for Economical Nebulization of Penicillin and Other Therapeutic Aerosols. Alvin L. Barach, Charles C. Rumsey Jr., Max Soroka and Dora Rader, New York State J. Med. **47**:1498 (July 1) 1947.

The authors have simplified the apparatus and the technic of home administration of penicillin mixtures. Three figures and a table illustrate the armamentarium and the method of procedure. In atomizing at home an automobile air pump worked by the foot may be used. The authors state that oral inhalation of 50,000 units of crystalline penicillin sodium produced blood levels in 12 patients with bronchopulmonary disease averaging 0.17 unit of penicillin per cubic centimeter of serum after one hour. While an oxygen tank is preferred, air pressure does not stuff the eustachian tubes as an aftermath of the treatment, which is likely to be the case with oxygen. Inhalations may be both nasal and oral and under "positive" or "negative" pressure, alternating as required.

The authors state that the equipment can be obtained from the Inhalational Equipment Company, 248 East One Hundred and Nineteenth Street, the Oxygen Equipment Manufacturing Corporation, 405 East Sixty-Second Street, in New York, or the Vaponefrin Company, 6812 Market Street, Upper Darby, Pa. The foot pump may also be obtained from these concerns. One should study the article in its entirety, however, before undertaking to administer the penicillin aerosol combination.

Voorhees, New York.

Miscellaneous

Management of Whooping Cough with Special Reference to Infants. Jerome L. Kohn and Alfred E. Fischer, Am. J. Dis. Child. **73**:663 (June) 1947.

Working in the Willard Parker Hospital, Department of Hospitals, New York, the authors give statistics for the year 1941 from the Census Bureau and state that there were 3,668 recorded deaths for that year. Ninety-four per cent of those who died were less than 3 years old, and 67 per cent were under 1 year. The disease begins in the nose and throat, but the diagnosis can seldom be made before "the whoop" is heard. In early cases, postnasal swab and culture or the "cough plate method" sometimes made the diagnosis. Blood counts also were helpful. The white cell count ran high—100,000 to 240,000—but had no prognostic significance. Because of anoxia due to secretion retained in the lower airways, tracheobronchial "direct tube" suction was used successfully, after which oxygen was inhaled, with 5 per cent carbon dioxide, through a funnel or under a tent. "Some patients showed cerebral injury, presumably as a result of anoxia." Penicillin was given to 9 patients, but conclusions were not definite. However, when both penicillin and the sulfonamide drugs were administered, improvement was noted. In outlining the treatment the following points were observed: (1) experienced nursing care; (2) oxygen inhalation; (3) aspiration of the tenacious secretions; (4) chemotherapy; (5) hydration and parenteral therapy; (6) special feeding technic; (7) use of hyperimmune and rabbit serums. Medicinal therapy was disappointing. Sedatives, antispasmodics, and expectorants accomplished little.

Voorhees, New York.

A Summary of Bronchoscopy in Diseases of the Chest. James M. Blake, New York State J. Med. **47**:1115 (May 15) 1947.

The material on which this article is based was studied in the Schenectady County Tuberculosis Hospital, Glenridge Sanatorium. Bronchoscopy is valuable

not only in diagnosis but, as an auxiliary, in treatment. Indications include: pulmonary hemorrhage, wheezing (suggestive of carcinoma), bronchial obstruction from whatever cause, unresolved pneumonia, nonspecific infections, foreign bodies, atelectasis (postoperative), pulmonary abscess, bronchiectasis, bronchogenic tumors, persistent cough and pulmonary or laryngeal tuberculosis. There seem to be few contraindications, and these would be self evident. Bronchoscopy may have to be performed repeatedly in pulmonary tuberculosis for the application of remedies (e. g., silver nitrate).

"Bronchoscopic examination should be considered where pulmonary symptoms . . . persist beyond the expected time. . . . As it is now performed in the modern bronchoscopic clinic . . . it is essentially an atraumatic procedure . . . which no longer can be looked upon as 'horrible.'" In these days of good roads from almost everywhere and fast automobiles it is possible for the rural patient to reach a bronchoscopist, since the number of these specialists is constantly increasing, and they are to be found in many smaller cities, wherever there is a hospital.

VOORHEES, New York.

BENADRYL: A SYNTHETIC ANTIHISTAMINE COMPOUND FOR THE SYMPTOMATIC TREATMENT OF ALLERGIC DISEASES. LAURENCE FARMER and HENRY SPICKSCHEN, New York State J. Med. **47**:1119 (May 15) 1947.

Working in the allergy clinic of Lenox Hill Hospital, the authors used "benadryl" (beta dimethylaminoethyl benzohydryl ether hydrochloride) in 108 allergic patients. Forty-three with pollinosis were given this drug in the summers of 1944 and 1945; 3 received it both years. Thirty-three had ocular or nasal symptoms or both, and 4 had additional bronchial disturbances. Five out of 10 of these had relief; 4 were not helped; in 1 the result was only "fair." Twenty-three had urticaria; 4 also had angioneurotic edema. The results here are considered "remarkable," since "only 1 was not benefited." A table shows the comparative effects. A positive diagnosis of allergy was insisted on, and "benadryl" was administered only "as needed." While certain cautions are enjoined, "it might be advisable to use Benadryl more regularly . . . in hay fever . . . two or three days before onset . . . and if well tolerated, 50 mg. three or four times a day could be taken during the Sp. pollinating period."

Untoward symptoms were dizziness and drowsiness, burning in mouth, throat or stomach, and red, sore, dry tongue, which annoyed some patients to the point of discontinuance. . . . The rationale of this treatment is based on the theory of histamine intoxication, and it is felt that a search for new derivatives of antihistaminic drugs should be made in the hope of doing away with objectionable and undesired results.

VOORHEES, New York.

PENICILLIN POISONING IN A CASE OF ACUTE STAPHYLOCOCCUS AUREUS HEMOLYTICUS INFECTION IN A HIP JOINT. NELSON W. CORNELL, New York State J. Med. **47**:1509 (July 1) 1947.

Although this is not a report of an otolaryngologic condition, it is well for otolaryngologists to know that "penicillin poisoning" is here reported, since they are often obliged to use this agent in large doses. After five days' usage of penicillin, 20,000 units being given every four hours, five times a day Cornell says, the supply gave out and a "different penicillin" was used in dosage of 25,000 units every four hours, six times a day. The following day there were generalized urticaria, paresis of the arms and the left foot, numbness and tingling of the

finger tips. The diagnosis was polyneuritis. The patient improved, went home but came back at two intervals with a recurrence of the infection in the same place, the right hip. "Heroic penicillin therapy was again instituted in the face of this serious complication." Eventually the man was dismissed with limited active and passive motion in the right hip joint and permanent foot drop on the left.

Comment by Reviewer: This calls for comment on the probable impurities of the second batch of penicillin! There was no trouble with the first batch over a five day period. In the medical profession the consensus was and still is that penicillin is almost harmless save for an occasional cutaneous rash, which promptly disappears when the drug is withheld. In the face of the fact that its life-saving value has been demonstrated in many thousands of cases reported in the literature, this instance of "poisoning" is open to challenge. Impure penicillin or some other unrecognized factor might explain the unfortunate outcome.

VOORHEES, New York.

ANTIHISTAMINE DRUGS IN ASTHMA AND HAY FEVER. DAVID LOUIS ENGELISHER, New York State J. Med. **47**:1696 (Aug. 1) 1947.

In his experience with 193 patients ranging from 3 to 79 years, the author found that such "newer remedies" as diphenhydramine hydrochloride ("benadryl hydrochloride") and N'pyridyl-N'benzyl-N-dimethylethylenediamine ("pyribenzamine") used for "a three-day trial" were fairly ineffective. Of 127 patients, roughly two thirds were unrelieved or their symptoms were aggravated because of the drying effect. Cough and asthma were observed in some patients who had not had such symptoms previously. Of the remaining third, about 9 per cent were improved. No patient who had severe asthma was convincingly relieved. Comparing the antihistamine agents with ephedrine, epinephrine, acetylsalicylic acid, phenobarbital, the iodides, etc., the author declares that the new drugs fail by far in effectiveness in allergy. Moreover, untoward symptoms of drowsiness, dizziness, headache, nausea and vomiting were often present. It is his opinion that although the antihistamine drugs may be of fair to striking value in a small percentage of cases of hay fever and asthma, the vast majority of the patients are not convincingly benefited; of those with asthma, the percentage is of negligible import, and such therapy does not "compare in value with our standard drugs and procedures."

VOORHEES, New York.

THE PHARMACOLOGY, PHYSIOLOGY AND CLINICAL EVALUATION OF THE NEW ANTIHISTAMINIC DRUGS (PYRIBENZAMINE AND BENADRYL). CARL E. ARBESMAN, New York State J. Med. **47**:1775 (Aug. 15) 1947.

This paper was read at the annual meeting of the New York State Medical Society, in Buffalo, May 7, 1947. The author works in the Buffalo General Hospital, the Buffalo Children's Hospital and the University of Buffalo School of Medicine. He states that the use of these newer drugs presents the "greatest advance in the therapeusis of allergy since the discovery of epinephrin in 1901." After giving some historical references to the progress that has been made, and reciting the chemistry and formulas of the antihistamine drugs, Arbesman describes clinical experiences in the treatment of seasonal allergic rhinitis, bronchial asthma and perennial allergic rhinitis. He says the new drugs are not ideal, but ventures the hope that they may yet be made so if research continues at the present pace. In summary, the author states that "pyribenzamine" (N'pyridyl-N'benzyl-N-dimethylethylenediamine) has been used by him since November 1944 in the treat-

ment of 800 patients with varying allergies. It relieved 78.5 per cent of 461 patients having allergic rhinitis of all types and also relieved 47 per cent of 159 patients with bronchial asthma. Of 199 persons showing urticaria, 78 per cent were benefited. "No serious side-effects have been noted in any patients, and many have had their daily dose for over two years." He concludes that "pyribenzamine" is more effective and less toxic than diphenhydramine hydrochloride ("benadryl hydrochloride"). "Antihistaminic drugs are palliative, and if permanent relief is to be obtained, the etiologic factors of allergic manifestations must still be determined and eliminated."

VOORHEES, New York.

MALIGNANT TUMORS OF THE SKIN IN MILITARY PERSONNEL. W. B. MACOMBER and F. X. PALETTA, Plast. & Reconstruct. Surg. **2**:234 (May) 1947.

The authors' description of their treatment of malignant new growths of the skin includes some cases of involvement of the nose. A squamous cell epithelioma of the ala nasi was excised, and reconstruction was carried out by rotating an adjacent nasolabial flap over the defect. When a tumor of the bridge was excised, the defect was repaired by using local wedge-shaped grafts drawn together or by full thickness skin grafts. If only the skin is involved, total excision can be done and the defect then covered by a full thickness graft from the supraclavicular area. When a growth of the nasal tip is excised, a visor flap is shifted downward from the bridge of the nose, and the denuded area is filled in with a full thickness graft. This prevents distortion of the contour of the tip. When the entire nose is involved, reconstruction is done with forehead flaps or neck tubes.

SELTZER, Philadelphia.

THE DANGERS OF LOCAL ANESTHESIA. VON REINHOLD KÄSER, Pract. oto-rhino-laryng. **8**:484, 1946.

The author discusses the dangers of unforeseen accidents that may occur as local anesthesia is induced for tonsillectomy. The first case discussed was that of a young woman who had recurrent attacks of tonsillitis and on whom tonsillectomy was to be performed. She received a preoperative injection of morphine sulfate and atropine sulfate. Forty-five minutes later about 20 cc. of a 0.5 per cent procaine hydrochloride solution containing 3 drops of a 1:1,000 epinephrine hydrochloride solution was injected into the mucosa of the pharynx. The patient suddenly became pale, with a marked spasm of the fingers and a profound collapse of the pulse. She was given 2 cc. of a 25 per cent nikethamide solution, after which she improved, and the tonsillectomy was completed. One and a half hours later she had another complete collapse. Emergency treatment with nikethamide, heat, and many other stimulants was administered. Convalescence continued to be stormy for about fourteen days, when she again had a complete collapse and died suddenly, of acute edema of the lungs.

The second patient was a well nourished man who had severe tonsillitis with marked cervical adenitis. After subsidence of the acute symptoms he was prepared for tonsillectomy. Morphine and atropine were given preoperatively. The pharynx was sprayed with a 2 per cent tetracaine hydrochloride solution, which was followed by a slow injection of 40 cc. of a 0.5 per cent procaine hydrochloride solution containing 10 drops of a 1:1,000 epinephrine hydrochloride solution. After fifteen minutes the patient suddenly became pale and then unconscious and foamed at the mouth. After considerable emergency stimulation there was no improvement in the condition, and within thirty minutes the patient died. Autopsy revealed status

thymicolymphaticus, dilatation of the right side of the heart, edema of the lungs and many areas of hemorrhage in the epicardium, the myocardium, the skin, and other tissues.

The third case was that of a young man who had had a severe sore throat. Tonsillectomy was indicated. Seven-tenths cubic centimeter of "pantopon" (a mixture of hydrochlorides of opium alkaloids) was administered on the operating table, for the patient appeared rather apprehensive. Forty-five minutes later the mucous membrane was painted with a 10 per cent cocaine hydrochloride solution. This was followed by an injection of 25 cc. of a solution containing procaine hydrochloride (0.2 per thousand) and "panthesine" (0.2 per cent) (a procaine derivative, methane sulfonate of *n*-diethyl-leucinolester of para-aminobenzoic acid) and 1 drop of a 1:1,000 epinephrine hydrochloride solution for every 10 cc. of the solution. A few minutes after the beginning of the induction of local anesthesia the patient became restless and gave curious answers. He was placed immediately in the Trendelenburg position but became pale and cyanotic. Suddenly, epileptiform seizures developed, with a clonic spasm of the entire body, a rapid pulse and irregular breathing. Within one half hour the patient died.

In view of these serious accidents the question arises as to the probability of allergic reactions to the cocaine group or the possibility of iodiosyncrasy to procaine. The psychic phase of the patient presents an extremely important problem. Status thymicolymphaticus must be considered in certain cases, particularly if epinephrine shock may be a factor. Whatever the problem may be, it seems that the rapid absorption of the anesthetic solution gives a dangerous hematic concentration of the drugs involved. This may occur through intramuscular or subcutaneous injections. To meet these accidents therapeutically, the author recommends that 5 to 10 cc. of a 10 per cent calcium chloride solution be injected intravenously. Barbital sodium and chloral hydrate, injected intravenously, are valuable to relieve the spastic condition.

PERSKY, Philadelphia.

News and Comment

THE KENFIELD MEMORIAL FUND

In 1937 a sum of money was subscribed in memory of Miss Coralie N. Kenfield of San Francisco, a teacher who was known throughout the United States for her high ideals and advanced methods of teaching lip reading. This money was placed in the Kenfield Memorial Fund. The interest provides a scholarship known as the Coralie Noyes Kenfield scholarship for Teachers' Training Courses for Teachers of Hard of Hearing Adults. (The scholarship offered in 1948 is $100.) The American Hearing Society is the trustee of the Kenfield Memorial Fund.

Applications will be considered from any hard of hearing teacher who is preparing to teach lip reading to hard of hearing adults and who lives in the United States and can meet the following requirements:

1. Personal characteristics necessary for successful teaching.

2. Ability to read lips as certified on examination by an approved instructor in lip reading.

3. A bachelor's degree; or two years of college work plus twelve semester hours of work in adult education, psychology of the handicapped, voice production and control, sight conservation or social service; or two years of successful experience in teaching in public or private schools plus twelve semester hours of work in adult education, psychology of the handicapped, voice production and control, social service or kindred subjects.

4. Professional training in lip reading distributed as follows: thirty clock hours of private instruction under an approved teacher of lip reading; or sixty clock hours of instruction in public school classes under an approved teacher of lip reading.

The winner of the scholarship may take the normal course from any normal training teacher or school or university in the United States offering a course acceptable to the teachers' committee of the American Hearing Society.

The applicant for the scholarship must be a prospective teacher. Applications from those who are teaching lip reading now cannot be considered. The scholarship must be used within one year after the granting of the award.

Applications must be filed between March 1 and May 1, 1948, with

Miss Rose V. Feilbach
2431 Fourteenth Street, N. W.
Washington 9, D. C.

THE FORTY-SIXTH FRENCH CONGRESS OF OTORHINOLARYNGOLOGY

The forty-sixth congress of the Société française d'Oto-Rhino-Laryngologie will be held in the Grand Amphitheatre of the Faculty of Medicine in Paris, October 18 to 23, under the presidency of Prof. L. Binet, dean of the Faculty of Medicine of Paris. Professor Terracol, of Montpellier, will present "Sinusitis in Infancy," and MM. J. Ramadier and C. Eyries, of Paris, "Surgical Treatment of Ozena."

During the congress, operatory seances will be held in the hospital services and an exposition of instruments and otorhinolaryngologic specialties will take place in the grand hall of the Faculty of Medicine.

Conferences have been arranged for the morning in the office of Professor Lemaitre in the Lariboisiere and for the afternoon in the small amphitheatre of the Faculty of Medicine. These conferences covering specialized subjects of medical or of surgical interest to the specialists will be given by Prof. G. Mauric, Prof. P. Mounier Kuhn, Prof. J. Despons, Dr. A. Mouchet, Dr. J. M. Le Mee, Dr M. Ombredanne and Dr. J. Tarneaud.

Excursions have been arranged for the week of October 18 to 23 for the congressmen and persons who accompany them. Arrangements for the excursions, as well as those for transportation and lodging in Paris, are assigned to the Wagons-Lits Co., 40 Rue de L'Arcade.

Further information may be procured from Dr. H. Flurin, Secretary General, 19 Avenue MacMahon, Paris, actually in Cauteret (Hautes Pyrenees), or Dr. H. Guillon, Asst. Secretary General, 6 Avenue MacMahon, Paris XVII.

GRADUATE INSTRUCTIONAL COURSE IN ALLERGY

A course of intensive graduate instruction in allergy will be presented by the American College of Allergists under the auspices of the University of Oregon Medical School, Portland, Ore., Nov. 8 to 12, 1948, inclusive. Hotel headquarters will be at the Heathman Hotel. The course includes all phases of the subject and will be presented by specialists in allergy. The treatment and management of the allergic patient will be considered in detail. There will be panel discussions and demonstrations of technics. Printed comprehensive abstracts of the lectures will be furnished the registrants. The course is available to members and nonmembers of the college. Applications should be placed with Dr. Fred W. Wittich, Secretary, The American College of Allergists, 423 La Salle Medical Building, Minneapolis 2, Minn. Programs will be mailed on request. The charge for the course is $75.00, payable at the registration desk.

MEETING OF AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF SURGERY

The sixth annual meeting of the American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery will be held at the Palmer House, Chicago, on Saturday, Oct. 9, 1948. Complete programs may be obtained by writing to the secretary, Dr. Norman N. Smith, 291 Whitney Avenue, New Haven 11, Conn.

Recent action by the society provides for associate and corresponding membership in addition to full fellowship. Complete details are available from the Secretary.

HOME STUDY COURSES

The Home Study Courses sponsored by the American Academy of Ophthalmology and Otolaryngology, in the basic sciences related to these two specialties, will be given again, beginning Sept. 1, 1948. Registrations must be completed before August 15. Detailed information may be secured from Dr. William L. Benedict, Executive Secretary, 100 First Avenue Building, Rochester, Minn.

HONORARY ELECTIONS

The Chicago Laryngological and Otological Society has recently announced the election to affiliate membership of Dr. Barry J. Anson, professor of anatomy, Northwestern University Medical School; Dr. Herbert Koepp-Baker, department of speech correction and audiology, University of Illinois, and Dr. Raymond Carhart, department of speech correction and audiology, Northwestern University. Affiliate membership is an honorary appointment granted by election to persons of high scientific attainment in fields related to otolaryngology.

Book Reviews

Hearing Aids: An Experimental Study of Design Objectives. By Hallowell Davis, project supervisor; S. S. Stevens, director, Psycho-Acoustic Laboratory; R. H. Nichols Jr., assistant director, Electro-Acoustic Laboratory; C. V. Hudgins; G. E. Peterson; R. J. Marquis; D. A. Ross. Price $2. Boston: Harvard University Press, 1947.

It is hard for an author, or authors, to please all of the readers. This is particularly true when the writing deals with material which is both technical and abstruse, or when, as happens often, the terminology is that of a branch of science unfamiliar to the reader. Thus, otologists have trouble in understanding the bulletins of the Bell Laboratories on acoustic problems. They have equal difficulty with the releases of the University of Iowa on the psychology of hearing.

Unfortunately, and fortunately, many specialists are mixed up in this matter of hearing—anatomists, physiologists, otologists, psychologists and acoustic engineers. Each speaks and writes in his own language.

This book is more than its title suggests, for it is a study of many of the frontier paths of the testing of hearing. In this respect it will probably be thoroughly digested by only a scant dozen otologists, those who are actively at work in this field.

The work is carefully done, and yet one wishes for a larger series than 18 cases to represent "a sample of the adult hard-of-hearing." This seems much too small a group from which to draw conclusions, and it is questionable whether 18 cases could represent all the degrees of disability occurring in various types of deafness. One recalls the confusions arising in regard to mixed types of deafness and in regard to the varying test responses obtained in patients with different degrees of otosclerosis, chronic progressive deafness, catarrhal deafness and senile deafness.

There are certain subjects dealt with concerning which there is still much difference of opinion. For example, many of the workers using speech hearing as a means of testing, completely discard meaningless test words. The technics of the authors vary considerably from those of others in the testing of intensity matching and in the use of noise as a dampener.

Another criticism is aimed at the use of terms that convey little meaning to the average reader—"spondiac," "compression amplification," "peak clipping" and "high tone preemphasis." Doubtless these are well selected labels, but they require a more simple elucidation than the text gives.

The tables used would be much more readily understood were keys provided to explain meaningless letters. One has to delve through the text in order to straighten the matter out.

Many items of importance are both reiterated and discovered in the studies made:

"Normal listeners understand 98 to 100 per cent of the words over a wide range of amplification when the frequency response is either flat or tilted up at the high-tone end. Maximum scores with low-tone emphasis are nearly but not quite as good."

"As optimum performance [of hearing aids] is approached the details of frequency pattern becomes less and less significant." "The most successful pattern is high tone emphasis." Yet the group (18 patients) as a whole slightly preferred the quality of a "flat amplification across the frequency range." "Low-tone

emphasis always gave poorer performance even for subjects with low-tone hearing loss." Reduction of the tones below 500 and above 4,000 cycles did not impair performance with the "Master Hearing Aid" (an experimental instrument designed to cover any aid requirements).

The authors believe that two or three models of hearing aids, differing primarily in acoustic gain, maximum power, size and cost. should be sufficient to cover the needs of all users. (There are now 16 aids acceptable to the Council on Physical Therapy of the American Medical Association.)

"A direct measurement of the hearing loss for speech is desirable." (This relates to the phonographic voice test.)

Bone conduction tests did not enter into the study because of the difficulties and uncertainties of this type of testing. "Bone-conduction thresholds are not standardized; bone-conduction receivers cannot adequately be calibrated by physical method; the amount of pressure and the position of application are both highly critical yet difficult to standardize; no simple adequate solution to the masking problem is available. Measurement of bone-conduction is still in the experimental stage for research purposes."

Hearing and Deafness: A Guide for Laymen. By Hallowell Davis, M.D. Pp. 471. Price $5. New York: Murray Hill Books, Inc., 1947.

This book should have been labeled "for intelligent laymen." It is quite inclusive and surely too intricate and technical for the average person interested in deafness. The chapter on anatomy and physiology is well done but belongs only in a scientific text or journal.

The various authorities have portrayed the many problems and facets of the hard of hearing in a convincing and interesting manner. Society as a whole is little concerned with its less fortunate members. Employers could take a leaf out of this compact volume and make excellent use of deaf and near-deaf people.

The wonderful work done by the Army and Navy hearing centers is brought to the fore. Civilian advancement of the teaching and the rehabilitation of the deaf must surely follow a somewhat similar pattern.

The portion devoted to the teaching of children, 18,000 completely deaf and about 1,500,000 hard of hearing, deserves a great deal of praise. Parents and teachers could both benefit from a perusal of this book; its implications, suggestions and explanations are invaluable.

Consummate patience is needed in all of this work if any one is to succeed at all in rehabilitating the deafened patient. Various types of vocational guidance are indicated. The best use of the hearing aid is described very explicitly. A great attempt is made to rid the handicapped patient of his inborn depression. He must be made to feel an integral part of all normal society—enabled to work in it and support himself and possibly a family—to enjoy himself among all people and not only in the company of those who are hard of hearing.

A rather new concept, "visible speech," thrown on a screen, opens up new avenues of hearing and interest, but it must be learned as any foreign language is.

The Bell Telephone Company and allied units are forever pushing research in the field of better hearing, and eventually the otologic patient benefits from their accurate testing apparatus, their compiled lists of words and sentences, and their tremendous improvements of the modern hearing aid.

There is no doubt but that any physician, whether specialist or physician in general practice, might enjoy scanning this little book and might add to it a few important items, and then pass it on to a very grateful child or adult afflicted with less than normal hearing.

The **Oculorotary Muscles.** By Richard G. Scobee, M.D. Price $8. St. Louis: C. V. Mosby Company, 1947.

Here is a well organized, well written, practical treatise on the extraocular muscles which it is a pleasure to read. The book contains thorough, but concise, sections on anatomy and physiology, heterophoria, heterotropia, diagnosis and treatment. Surgical technics, however, are not included, although general surgical principles are discussed.

While the authorities are cited throughout, the citations are far from being mere repetitions of orthodox opinions. The author has a stimulating array of original thought and research with which to enliven his writing.

The concept of lateral phoria as a single innervational entity depending on the state of tonicity of a convergence center is definitely appealing by reason of its simplicity and plausibility. Chapters on hyperphoria and cyclophoria deserve to be well studied by the refractionist. Hyperphoria is presented from the standpoint of White, who stated, in 1932, that it was caused in 98 per cent of the cases by a weakness of one or more of the elevator or depressor muscles.

Extremely useful and illuminating comments on all matters pertaining to the extraocular muscles are repeatedly encountered in the course of reading. Decidedly this is a valuable monograph for any one concerned with the subject.

Otovestibular Symptomatology and Intracranial Tumors. By Dr. Ausano Della Vedova, otologist of Victor Emanuel III, Neurological Institute, Milan. Pp. 213, with 30 illustrations. Paper. Price 75 lire. Milan, Italy: A. Saita, Via Simone d'orsenigo, 2. Pozzi, 1942.

The author presents a thorough description of the anatomic and physiologic aspects of the internal ear and eighth nerve, and of the latter's intracranial connections. He describes with deep insight the pathologic appearance and symptoms of labyrinthine and intracranial lesions, and makes a laudable attempt to demonstrate the relationship between neurology and otology in this field.

He gives factual and theoretic explanations of the various tests and reactions which aid in the diagnosis of these lesions. He gives an excellent method of examination, including hearing, rotary, caloric—both unilateral and simultaneously bilateral—and galvanic tests.

He includes a detailed description of 105 intracranial lesions, all of which came to operation or to autopsy, corroborating or correcting his experimental findings.

These lesions were: 7 lesions of the brain stem; 11 of the eighth nerve; 9 of the cerebellum, in the midline; 18 of the cerebellum, lateralized to the right or the left; 7 of the posterior fossae; 12 supratentorial lesions in the midline, and 41 supratentorial lesions lateralized to the right or the left.

Notice

Requests from libraries, medical societies and individual physicians in various devastated countries are being received almost daily. If physicians have extra copies of medical journals or copies for which they have no further use, it will be greatly appreciated if they will send them to the headquarters of the Association for distribution abroad.

Directory of Otolaryngologic Societies *

INTERNATIONAL

Second Pan-American Congress of Oto-Rhino-Laryngology and Bronchoesophagology

President: Prof. Justo Alonso.
Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia 3.
Place: Montevideo. Time: January 1950.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Henry B. Orton, 224 Delavan Ave., Newark, N. J.
Secretary: Dr. J. Milton Robb, 1553 Woodward Ave., Detroit, Mich.
Place: Atlantic City, N. J. Time: June 6-10, 1949.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5.
President-Elect: Dr. C. H. McCaskey, 20 N. Meridian St., Indianapolis, Ind.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100–1st Ave. Bldg., Rochester, Minn.

American Broncho-Esophagological Association

President: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore.

American Laryngological Association

President: Dr. Frederick T. Hill, 177 Main St., Waterville, Maine.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.

SECTIONS:

Eastern.—Chairman; Dr. Harold G. Tobey, 403 Commonwealth Ave., Boston.
Southern.—Chairman: Dr. Watt W. Eagle, Duke University, Durham, N. C.
Middle.—Chairman: Dr. Dean M. Lierle, University Hospital, Iowa City.
Western.—Chairman: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.

American Otological Society

President: Dr. Marvin F. Jones, 721 E. 60th St., New York.
Secretary: Dr. Gordon D. Hoople, 713 E. Genesee St., Syracuse, N. Y.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Jacob Daley, 104 E. 40th St., New York 16.
Secretary: Dr. Norman N. Smith, 291 Whitney Ave., New Haven, Conn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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